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DIAGNOSTIC VALUE OF DEFECTS IN THE VISUAL FIELDS AND OTHER OCULAR DISTURBANCES

ASSOCIATED WITH SUPRATENTORIAL TUMORS OF THE BRAIN

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This communication is based on a clinical and anatomic survey of 171 verified supratentorial tumors of the brain studied in the neurologic service and in the neuropathologic laboratory of the Mount Sinai Hospital. The investigation was undertaken in an attempt to answer the following questions:

1. Of what assistance are studies of the visual fields and observations on other ocular disturbances, such as diminished acuity of vision, papilledema, optic atrophy, alterations of the pupillary reflex and ocular palsies, in the localization of brain tumor?

2. How frequently do the observations at operation or on postmortem examination corroborate the clinical diagnosis, particularly when the latter is based on defects in the visual fields?

3. What factors aside from the location of the tumor are responsible for the presence or absence of the commonly characteristic ocular disturbances?

For the needs of this study the topographic grouping of tumors of the brain in common usage was found inadequate. The accurate correlation of the clinical manifestations with the precise location of the tumor in question called for a more elaborate classification of supratentorial tumors in accordance with a more exact outline of their frequent sites as revealed by a careful anatomic study of our material (figs. 1, 6, 9, 10, 24, 25 and 30; table 1). For the same reason it was found desirable to subgroup the tumors as encapsulated (A) and infiltrating (B).

Awarded the Lucien Howe Gold Medal in Ophthalmology.

From the Neurological Service of Dr. Israel Strauss and the Division of Laboratories, Mount Sinai Hospital.

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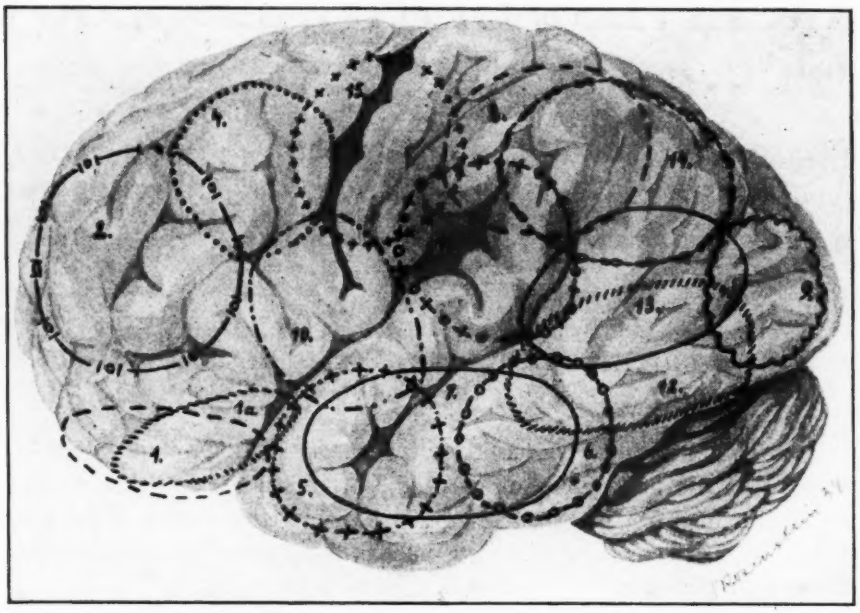

Fig. 1.—Frequent sites of tumor of the brain: (1) subfrontal, (2) prefrontal, (4) frontal, (5) temporal anterior, (6) temporal posterior, (7) temporal lobar, (8) parietal, (9) occipital, (10) temporofrontal, (11) temporoparietal, (12) temporo-occipital, (13) temporo-occipitoparietal, (14) occipitoparietal and (15) frontoparietal.

TABLE 1.—*Grouping of Supratentorial Tumors*

	A	B	C
1. Subfrontal	Encapsulated	Infiltrating	
2. Prefrontal	Encapsulated	Infiltrating	
3. Prefrontal interlobar	Encapsulated	Infiltrating	
4. Frontal	Encapsulated	Infiltrating	
Frontal interlobar	Encapsulated		
5. Temporal anterior	Encapsulated	Infiltrating	
6. Temporal posterior	Encapsulated	Infiltrating	
7. Temporal lobar	Encapsulated	Infiltrating	
8. Parietal	Encapsulated	Infiltrating	
9. Occipital	Encapsulated	Infiltrating	
10. Temporofrontal	Encapsulated	Infiltrating	
11. Temporoparietal	Encapsulated	Infiltrating	
12. Temporo-occipital	Encapsulated	Infiltrating	
13. Temporo-occipitoparietal	Encapsulated	Infiltrating	
14. Occipitoparietal	Encapsulated	Infiltrating	
15. Frontoparietal	Encapsulated	Infiltrating	
16. Intraventricular (lateral ventricles)			
17. Basal ganglions			
18. Interpeduncular	Suprasellar meningioma	Craniopharyngioma	Pituitary adenoma
19. Third ventricle			
20. Diffuse hemispheric			

In assembling our observations, to avoid redundance, we have refrained from giving lengthy descriptions of the individual cases, believing that the purpose of this contribution will be fully served by presenting in table form (tables 2 to 20) the salient points in all of the cases to be recorded.

SUMMARY AND EVALUATION OF THE RECORDED OBSERVATIONS

VISUAL FIELDS

A. Predictable defects in the visual fields were noted in the following groups:

Group 1 A. Subfrontal Encapsulated Tumors (5 cases; table 2 and figs. 2 *a-b* and 6).—Here, owing to the tumor's direct implication of the anterior (prechiasmal or chiasmal) segments of the optic pathways in varying degree, the defects in the visual fields were similarly irregular and inconstant. At times bitemporal field defects of the type frequently seen in cases of pituitary tumor were present, or the defects assumed the character of an asymmetrical bitemporal hemianopia; often one eye was totally blind, while the other showed temporal hemianopia. Then, again, vision in both eyes might be reduced to such an extent as to make reliable studies of the visual fields impossible.

Group 6 A. Temporal Posterior Encapsulated Tumors (6 cases; table 7 and fig. 2 *i-j*).—The disturbances in the visual fields in this group were inconstant and offered no characteristic defects. In only 1 case was there a contralateral temporal quadrantic defect, but there the site and extent of the tumor could be determined only during operation. In this instance the field defect may perhaps be regarded as the beginning of homonymous hemianopia, which, as will be noted later, may often develop in this fashion.

Group 6 B. Temporal Posterior Infiltrating Tumors (6 cases; table 7 and figs. 3 *a-b* and 14).—In this group, with good cooperation on the part of the patients, typical either quadrantic or hemianopic homonymous defects were almost always obtained. Failure to obtain characteristic homonymous field defects was commonly due to the intellectual depreciation of the patient, caused, not unlikely, by the invasive character of the lesion. It is rather significant that a homonymous field defect in association with an encapsulated tumor in this location was the exception (see group 6 A).

Group 7 B. Temporal Lobar Infiltrating Tumors (6 cases; table 8 and figs. 3 *c-d* and 15).—Here, too, homonymous hemianopic or quadrantic defects would have been a constant finding had not the mental disability of the patients frequently made studies of the visual fields impossible.

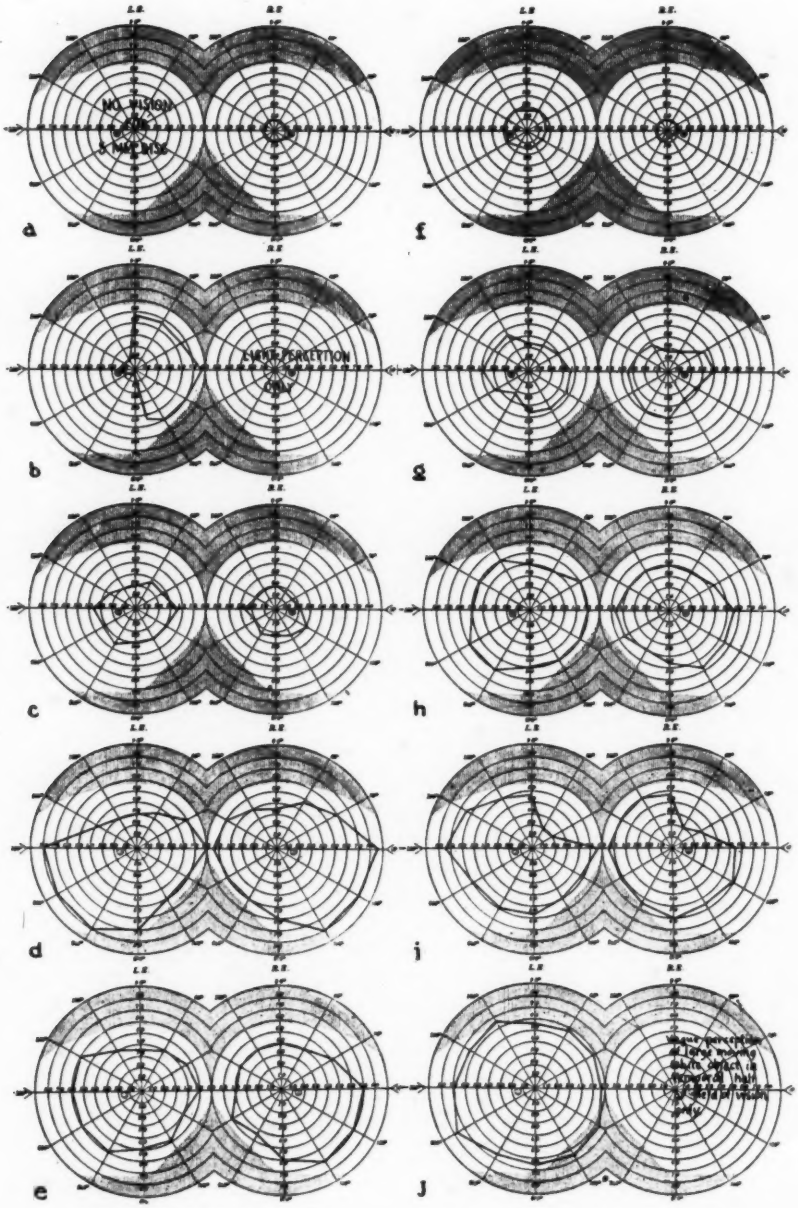

Fig. 2.—Visual fields: (*a*) and (*b*) cases 1 and 4, group 1A, table 2; (*c*) case 5, group 2B, table 3; (*d*) case 1, group 3A, table 4; (*e*) case 1, group 3B, table 4; (*f*) case 1, group 4A, table 5; (*g*) and (*h*) cases 4 and 6, group 4B, table 5; (*i*) and (*j*) cases 3 and 5, group 6A, table 7.

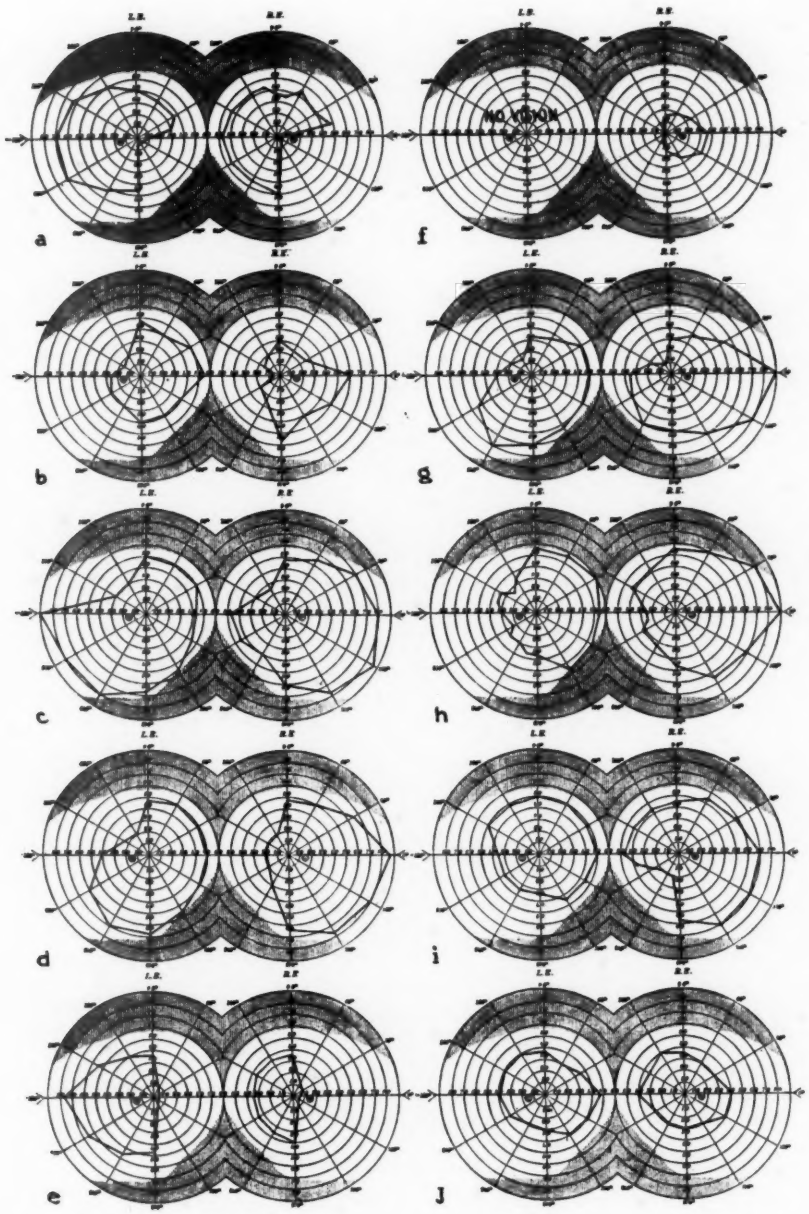

Fig. 3.—Visual fields: (*a*) and (*b*) cases 2 and 6, group 6B, table 7; (*c*) and (*d*) cases 5 and 6, group 7B, table 8; (*e*) (*f*) and (*g*) cases 1, 4 and 5, group 8A, table 9; (*h*), (*i*) and (*j*) cases 8, 9 and 11, group 10B, table 11.

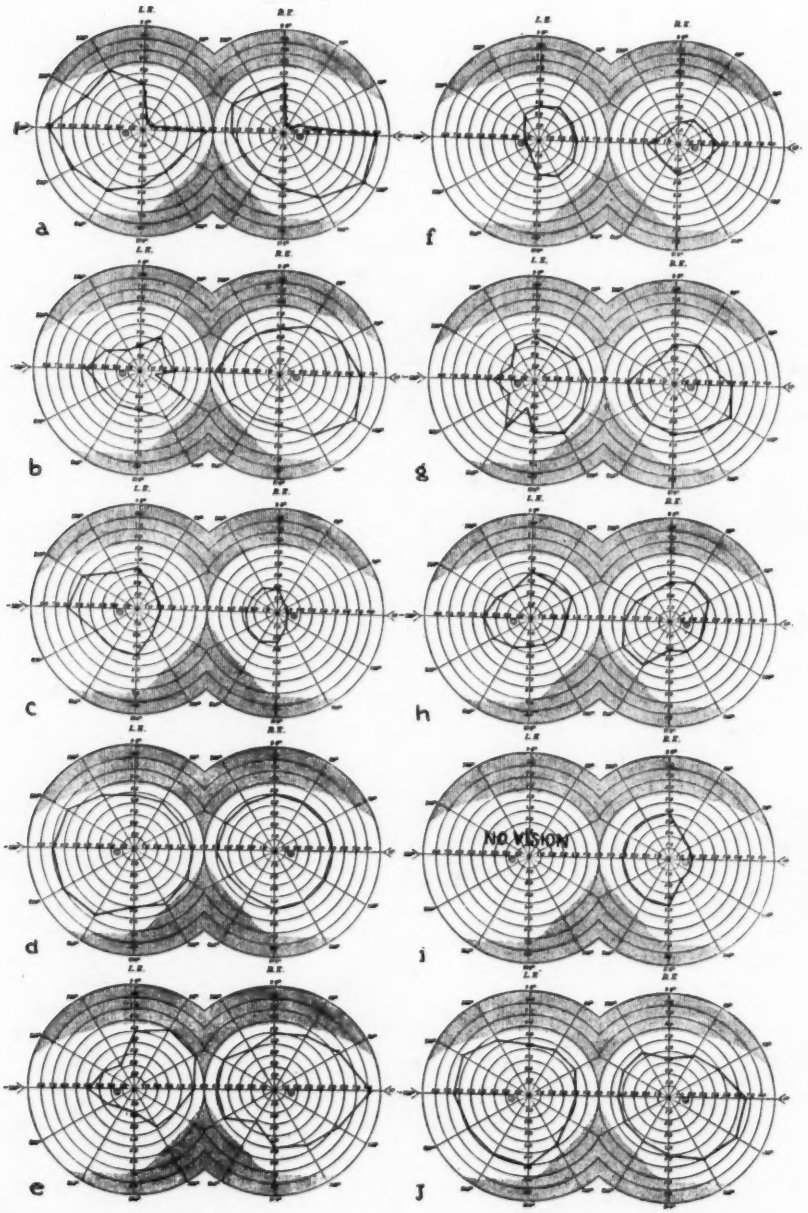

Fig. 4.—Visual fields: (*a*), (*b*), (*c*) and (*d*) cases 3, 6, 10 and 11, group 11B, table 12; (*e*) case 1, group 14A, table 14; (*f*) case 6, group 14B, table 14; (*g*) and (*h*) cases 3 and 4, group 15B, table 15; (*i*) case 1, group 18A, table 18; (*j*) case 6, group 18B, table 18.

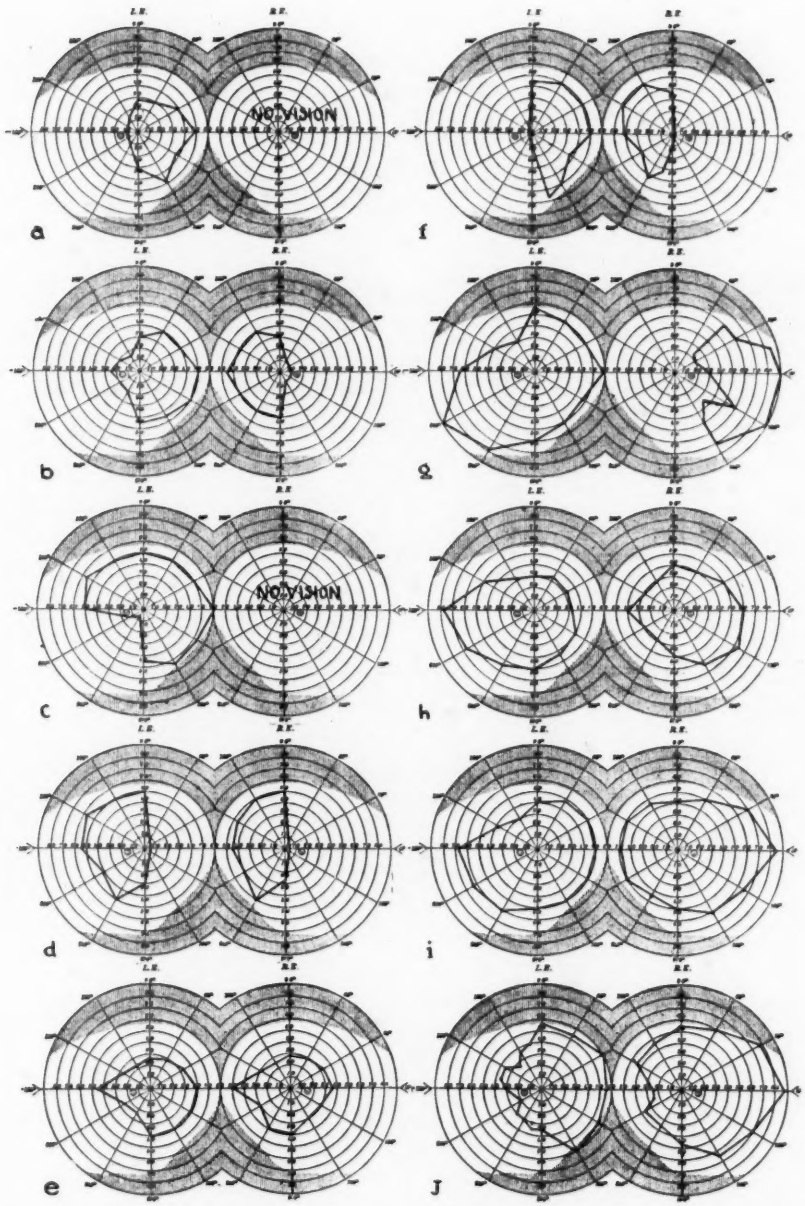

Fig. 5.—(*a*), (*b*), (*c*), (*d*), (*e*), (*f*) *and* (*g*) cases, 1, 2, 3, 5, 6, 7 and 8, group 18C, table 18; (*h*) case 6, group 20, table 20; (*i*) and (*j*) case 8, group 10B, table 11.

TABLE 2.—Sub

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 1A. Encapsulated (fig. 6)						
1	7987	—	O. D., extreme contraction with only a very small central field left intact (fig. 2a) O. S., no vision	O. D., 3 diopters O. S., 3 diopters	O. D., fingers at 20 ft. (6 meters) O. S., fingers at 2 ft. (61 cm.)	O. D., none O. S., pallor (secondary atrophy?)
2	7821	—	Not obtained because of amaurosis	Bilateral indistinct margins	O. D., almost complete blindness O. S., total blindness	O. D., none O. S., early primary
3	3973	—	Intact (grossly)	Slight bilateral superimposed	Good (grossly)	Bilateral low grade primary
4	X406	—	O. D., no vision O. S., temporal hemianopia (fig. 2b)	None	O. D., perception of light O. S., not observed	Bilateral primary
5	36102S§	—	O. D., defect in lower temporal quadrant O. S., temporal hemianopia	O. D., 2 diopters O. S., 1.5 diopters	O. D., 20/30 O. S., perception of light	Bilateral postneuritic L>R

* R equals right; L, left.
† The preoperative period is the length of time elapsed between the appearance of the early subjective complaints which usher in the clinical course and the making of a diagnosis and taking of surgical steps.
§ S following a number indicates that the case was verified only surgically.

SUMMARY

An analysis of the visual disturbances in the 5 cases in this group shows that the most characteristic findings in the presence of these subfrontal encapsulated tumors were: (1) a marked reduction in visual acuity, often to the extent of total blindness in the more involved eye; (2) primary optic atrophy in various stages of development; (3) defects in the visual fields which were difficult to obtain because of the advanced loss of vision; when they were obtained, occasionally a unilateral temporal field defect was found. All other ocular manifestations, such as inequality of the pupils and poor reaction of the pupils to light, were proportional to the depreciation of vision. The difference in palpebral fissures was readily accounted for by a difference in the facial innervation. In figure 6 four tumors in this group are drawn to scale, with indications as to their location, relative size and relation to the optic nerves. The preoperative period was variable; the shortest one was eleven weeks and the longest nine years.

rontal Tumors

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period†	Comment
Equal	Poor	Bilateral external rectus palsy	(P), R>L* (F), right (slight)	Impaired convergence; bilateral exophthalmos R>L	11 weeks	Subjective diminution of vision 4 weeks; history of transient internal squint 3 weeks
L>R	R, poor L, fixed	Intact	(P), R>L (F), right	None	7 months	Subjective diminution of vision on left 5 weeks
Not observed	Poor	Intact	(P), R>L (F), right	None	8 years	Patient disoriented and uncooperative; subjective impairment of vision 3 years; history of diplopia 6 months
R>L	R, paradoxical L, good	Intact	(P), equal (F), none	Right eye lags on convergence; occasional lateral nystagmus	9 years	History of blindness on right 3 years; failing vision on left 1 year
L>R	R, good L, paradoxical	Intact	(P), equal (F), none	Bilateral exophthalmos R>L	22 months	Slight improvement after 2 years

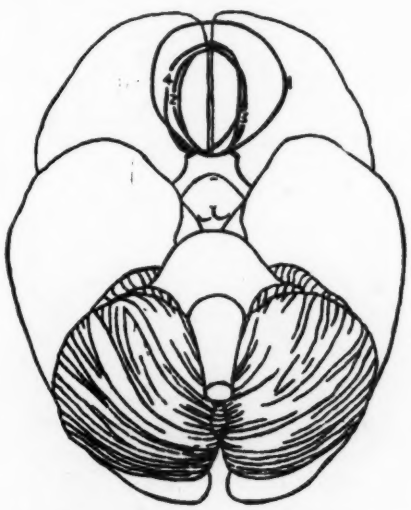

Fig. 6.—Location of subfrontal encapsulated tumors (group 1A, table 2).

TABLE 3.—Pr

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 2A. Encapsulated (fig. 7)						
1	6389	R	Slight contraction of upper temporal fields	O. D., 6 diopters O. S., 6 diopters	Very poor	None
2	3903	R	Not observed	None	Not observed	None
Group 2B. Infiltrating (fig. 8)						
1	8580	L	Not observed	None	Good (grossly)	None
2	6957	L	Not observed	Bilateral R>L	Not observed	None
3	5339	L	Not observed	O. D., 4 diopters O. S., 5 diopters	Not observed	None
4	3940	L	Intact (grossly)	O. D., 2 diopters O. S., 4 diopters	Good (grossly)	None
5	X382	L	Marked bilateral uniform contraction (fig. 2c)	Bilateral, early	Good (grossly)	None
6	47942S	L	Intact	Bilateral blurring	O. D., 15/40 O. S., 15/40	None
7	35233S	L	Intact	O. D., 3 diopters O. S., 3 diopters	Good	None
8	7388	R	Intact	O. D., 4 diopters O. S., 2 diopters	O. D., 10/70 O. S., 10/70	None
9	7117	R	Not observed	None	Not observed	None
10	4086	R	Not observed	None	Good	None
11	45370S	R	Intact	O. D., blurred O. S., 1 diopter	O. D., 10/15 O. S., 10/15	None

SUMMARY

In group 2 A the cases are too few to allow us to draw any conclusions. Highly unusual, however, was the presence in case 1 of a very high papilledema with the tumor situated far forward. Similarly unusual was the bilateral external rectus paresis found in both cases. The bilateral exophthalmos was in keeping with the location of the lesion. Of note is the absence of optic atrophy and the fact that in case 2 the larger pupil was on the side opposite the tumor.

In the prefrontal area the infiltrating type of tumor (group 2 B) is apparently more frequent than the encapsulated. In this group the mental change, which in itself is an important diagnostic sign, was a deterrent in obtaining dependable visual fields. However, with good cooperation on the part of the patient, perimetry revealed fields with no distinctive features except for an occasional uniform contraction as in case 5. Papilledema was not common; when found, it varied in degree, being sometimes as high as 4 or 5 diopters. The inequality of the papilledema was highly irregular, the more marked swelling bearing no definite relationship to the side in which the tumor was located. The visual acuity occasionally showed some reduction. In none of the cases was there evidence of optic atrophy. In some instances inequality of the pupils was found; here, too, no constant relationship could be noted between the contracted or dilated pupil and the side in which the lesion was found. Involvement of the extrinsic muscles was uncommon. Conjugate deviation of the eyes to the side opposite to the location of the lesion was occasionally present (2 cases).

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	Good	Bilateral external rectus palsy	(P), L>R (F), left	None	6 months	History of impaired vision 6 months; death 7 hours after encephalography
L>R	Poor	Bilateral external rectus palsy	Not observed	Bilateral exophthalmos	4 months	Mental changes; patient uncooperative
Equal	Good	Intact	(P), L>R (F), left	Conjugate deviation to right, horizontal nystagmus during convulsions	2 weeks	Death during status epilepticus
Equal	Good	Not observed	(P), equal	None	1 year	Mental changes; patient uncooperative
Equal	Good	Intact	(F), right	Slight convergence squint	7 weeks	
R>L	Good	Left external rectus palsy	Not observed	Occasional nystagmus on gazing to left	7 weeks	Patient aphasic, uncooperative
L>R	Poor	Intact	(P), R>L (F), right	None	14 months	Mental changes, poor cooperation
Equal	Good	Intact	(P), R>L (F), right	None	3 years	History of diplopia 3 weeks; improvement 6 weeks after operation
Equal	Good	Intact	(P), equal (F), none	Impaired convergence	4 years	Three craniotomies in 3 years; death 1 year after last craniotomy
L>R	Good	Intact	(P), equal	Nystagmus on gazing to right	2 months	Slight impairment of vision few days
R>L	Good	Intact	(P), equal	None	4 months	Patient bedridden; rapid decline
L>R	Good	Bilateral external rectus palsy; right internal rectus weakness	(P), equal; bilateral pseudoptosis	Conjugate deviation to left, horizontal nystagmus during convulsive attack	3 years	History of gradual impairment of vision 3 years; patient aphasic, uncooperative
Equal	Good	Intact	(P), equal (F), none	None	8 weeks	Three operations in 2 years; improvement 17 months after operation

Figure 7

Figure 8

Fig. 7.—Location of prefrontal encapsulated tumors (group 2A, table 3).

Fig. 8.—Location of prefrontal infiltrating tumors (group 2B, table 3).

TABLE 4.—*Pre*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 3A. Interlobar Encapsulated (fig. 9)						
1	8677	L	Contraction of upper half of left fields (fig. 2d)	O. D., 1 diopter O. S., 1 diopter	O. D., 15/30 O. S., 15/30	None
2	8778	R	Intact (grossly)	O. D., 2 diopters O. S., 1 diopter	Good	None
3	44229S	R	O. D., small field in temporal half preserved (see last column) O. S., intact	O. D., none O. S., slight blurring	O. D., perception of light O. S., 20/30	None
Group 3B. Bilobar Infiltrating (fig. 10)						
1	6703	—	Slight uniform peripheral contraction (fig. 2e)	O. D., 1.5 diopter O. S., 2 diopters	Good	None
2	5443	—	Not observed	None	Not observed	None
3	4684	—	Not observed	None	Not observed	None

SUMMARY

The prefrontal interlobar encapsulated and bilobar infiltrating tumors produced no distinctive ocular defects. In the majority of instances the disks were within normal limits; when papilledema was encountered, it was low—not higher than 2 diopters. In none of the cases was optic atrophy found. Striking was the difference in the preoperative period between the encapsulated tumors (very long) and the infiltrating (very short).

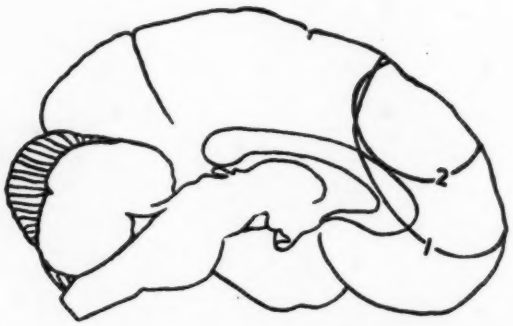

Fig. 9.—Location of prefrontal interlobar encapsulated tumors (group 3A, table 4).

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
L>R	Poor	Intact	(P), equal	None	4 years	History of visual difficulty 1 year, corrected by glasses
L>R	Good	Intact	(P), L>R (F), left	None	7 months	Mental changes; patient uncooperative
Equal	R, poor L, good	Right internal rectus weakness	Not observed	Right divergent squint	20 years	Old posttraumatic chorioretinitis on right; improvement 20 months postoperative
R>L	Good	Intact	(P), R>L (F), left	Impaired convergence	9 weeks	History of attacks of diplopia on gazing to left; death 24 hours after trephine exploration
Equal	Poor	Intact	Not observed	None	2 weeks	Mental changes; patient uncooperative; death before operation
R>L	Poor	Intact	(P), equal	None	10 weeks	Patient admitted in stupor; death before operation

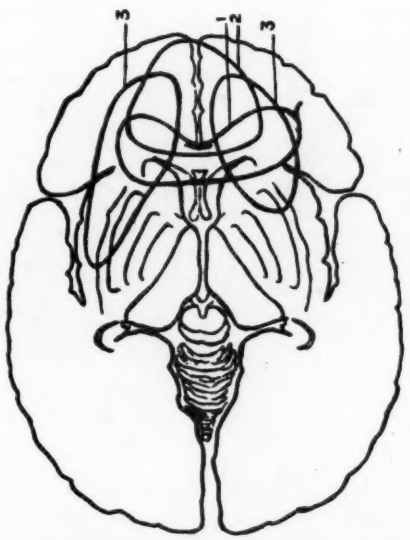

Fig. 10.—Location of prefrontal bilobar infiltrating tumors (group 3B, table 4).

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 4A. Encapsulated (fig. 11)						
1	X300	L	Marked contraction (only small central field of vision left) (fig. 2f)	O. D., 2 diopters O. S., none	O. D., good (grossly) O. S., very poor	O. D., none O. S., faint temporal pallor
2	32171S	L	Intact (grossly)	Very slight, bilateral	Not observed	None
3	7435	R	Not observed	O. D., 2 diopters O. S., none	O. D., 10/200 O. S., 10/200	O. D., none O. S., early primary
4	5674	R	Intact (grossly)	O. D., 4 diopters O. S., 4 diopters	O. D., 15/100 O. S., 15/100	None
5	40576S (X393)	R	Intact (grossly)	O. D., 3 diopters O. S., 2 diopters	Not observed	None
6	36018S	R	Intact	O. D., 4.5 diopters O. S., 4 diopters	O. D., 20/20 O. S., 20/20	None
Group 4A[1]. Interlobar Encapsulated (Parasagittal)						
1	34856S	L	Intact (grossly)	O. D., 2 diopters O. S., 2 diopters	Good (grossly)	None
2	33805S	L	Intact	O. D., 1 diopter O. S., 1.5 diopters	O. D., 15/20 O. S., 20/20	None
3	35491S	R	Intact	O. D., 2.5 diopters O. S., 2 diopters	Good (grossly)	None
Group 4B. Infiltrating (fig. 12)						
1	8334	L	Intact	O. D., 2 diopters O. S., 1.5 diopters	Impaired (grossly)	None
2	6272	L	Not observed	O. D., blurred O. S., 1 diopter	Not observed	None
3	4547	L	Not observed	Bilateral blurring	Not observed	None
4	27586S	L	Marked concentric contraction (fig. 2g)	O. D., 6 diopters O. S., 6 diopters	Good (grossly)	None
5	7837	R	Not observed	O. D., blurred nasal margins O. S., none	Not observed	None
6	6819	R	Slight uniform contraction (fig. 2h)	O. D., 2 diopters O. S., 2 diopters	Not observed	None
7	5240	R	Not observed	Bilateral blurring R>L	Not observed	None

SUMMARY

The frontal encapsulated tumors, with a single exception, produced no defects in the visual fields. In the excepted case there was marked contraction of the fields. Papilledema was present in all cases and varied from very slight in case 2 to 4 and 4.5 diopters in other cases. The visual acuity in 3 cases was markedly reduced; in 1 it was intact and in 2 not observed. A faint temporal pallor in one eye was the only indication of optic atrophy found in case 1.

The inequality of the pupils was in no relation to the site of the lesion, and the ocular muscles were all intact. Impaired convergence was present in only 1 case. In general, in a group of tumors of this type little aid can be obtained from the study of ocular manifestations as localizing signs.

In the 3 cases in group 4 A[1] only surgical descriptions of the location and size of the tumor are available. These descriptions are, however, sufficiently

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	R, good L, poor	Intact	(P), R>L (F), right	Impaired convergence	17 months	Kennedy syndrome ?
Not observed	Good	Intact	Not observed	None	6 years	
Equal	Good	Intact	(P), equal	None	3 months	Myopia; patient uncooperative; mental changes
R>L	Not observed	Intact	(P), L>R (F), left	Mild exophthalmos	8 months	History of blurred vision, bright flashes, blind spots 8 months
Equal	Good	Intact	(P), L>R (F), left	None	2 years	
L>R	Good	Intact	(P), L>R (F), left	Divergent squint; occasional nystagmus on right lateral gaze	7 months	Two operations in 3 years
Equal	Good	Intact	(P), equal (F), none	Occasional nystagmoid jerks; impaired convergence	9 months	Improvement for 3½ years; final result not known
Equal	Good	Intact	Not observed	None	2 years	Slight improvement 14 months
Equal	Good	Intact	Not observed	Occasional lateral nystagmus	1 year	History of blurred vision 8 weeks; patient unimproved 3 months after operation
L>R	Good	Intact	(P), equal	None	7 weeks	History of blurred vision 1 week
Not observed	Poor	Right external rectus palsy	(P), equal	None	2½ years	Mental changes; patient uncooperative
Equal	Not observed	Not observed	Not observed	Not observed	8 months	Patient aphasic; convulsions; emergency craniotomy
R>L	R, poor L, good	Intact	Not observed	Occasional nystagmus on lateral gaze	9 weeks	
Not observed	Not observed	Intact	(P), L>R (F), left	None	3 months	Progressive decline; death 6 days after ventriculography
Unequal	Poor	Bilateral external rectus palsy	(P), R>L (F), present, side not observed	Occasional nystagmus on lateral gaze	7 months	None
Equal	Good	Left superior rectus weakness; bilateral external rectus palsy	Not observed	None	10 weeks	Mental changes; stupor; death before operation

complete to place the cases in this group. No characteristic ocular manifestations were obtained.

Infiltrating tumors (group 4 B) appear to be somewhat more common in the frontal lobe than encapsulated tumors. It is perhaps their infiltrating character that is responsible for the greater anatomic disruption that causes more advanced intellectual depreciation than is seen in patients with encapsulated tumors. Mental deterioration made it impossible in a number of these cases to obtain reliable visual fields. In those in which they were obtained, nothing significant could be found. The papilledema which was present in all cases was exceedingly variable. In case 4 it reached the degree of 6 diopters. The visual acuity could not be observed. In none of the cases was there optic atrophy. The pupillary inequalities and the casual deviation in the action of the ocular muscles were inconstant and of little diagnostic value. In no instance were there findings which could be interpreted as the Kennedy syndrome.

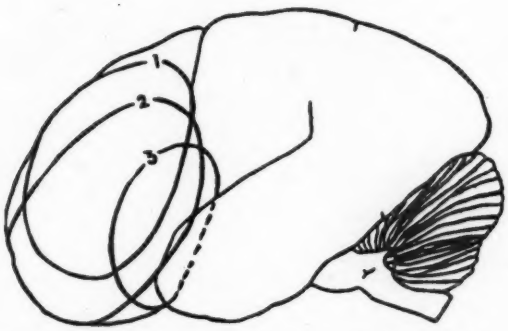

Fig. 11.—Location of frontal encapsulated tumors (group 4A, table 5).

TABLE 6—*Anterior*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 5B.	Anterior	Infiltrating	(fig. 13)			
1	7884	L	Not observed	O. D., none O. S., blurred	Not observed	None
2	5968	L	Intact	O. D., slight blurring on nasal side O. S., none	Not observed	None
3	X331	L	Intact	Bilateral, slight	O. D., 10/10 O. S., 10/10	None
4	7777	R	Not observed	Bilateral, advanced	Fingers not recognized at 1½ feet (48 cm.) from eyes	None
5	5748	R	Intact (grossly)	Bilateral blurring	Not observed	None

SUMMARY OF TABLE 6

An anterior temporal lobe tumor presents the most difficult problem of localization unless the lesion is on the left side where speech defects may point to its site. The absence of defects in the visual fields distinguishes this group from other tumors of the temporal lobe. This, however, in itself, is of little diagnostic aid in lesions of the right side. The papilledema was usually slight, visual acuity was unimpaired, and there was no optic atrophy. Of some significance was the slight limitation of the upward gaze.

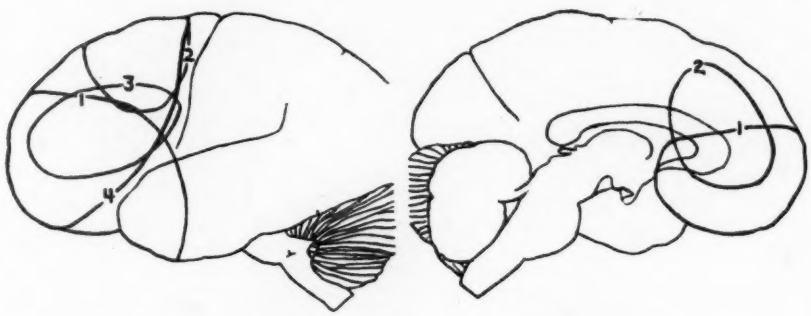

Fig. 12.—Location of frontal infiltrating tumors (group 4B, table 5).

Temporal Lobe Tumors

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	Good	Intact	(P), R>L (F), right	None	8 weeks	Mental deterioration; semi-stupor; patient aphasic
Equal	Good	Intact	(P), R>L (F), right	None	6 weeks	
R>L	Good	Intact	Not observed	None	1½ years	
L>R	Poor	Intact	(P), R>L	Slight limitation of upward gaze L>R; occasional nystagmus on right lateral gaze	6 months	History of diminution of vision 2 weeks
Equal	Good	Left external rectus palsy	Not observed	Slight limitation of upward gaze; occasional nystagmus on lateral gaze	3 months	Poor cooperation

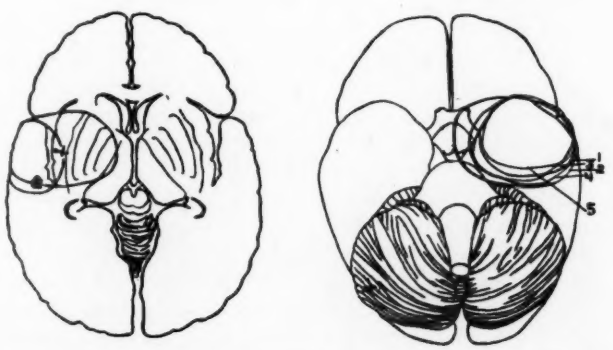

Fig. 13.—Location of anterior temporal infiltrating tumors (group 5B, table 6).

TABLE 7.—*Posterior*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 6A. Encapsulated						
1	7465	L	O. D., uniform contraction O. S., no vision	O. D., 1 diopter O. S., none	O. D., 15/15 O. S., amaurotic	O. D., none O. S., primary
2	6258	L	Not observed	O. D., early O. S., 2.5 diopters	Not observed	None
3	44681S	L	Defect in right upper temporal quadrant (fig. 2i)	None	O. D., 15/20 O. S., 15/30	None
4	44579S	L	Intact	None	O. D., good O. S., poor	None
5	35334S	R	O. D., vague perception of large moving objects O. S., intact (fig. 2j)	O. D., 2.5 diopters O. S., 2.5 diopters	O. D., vague perception of light or large moving objects O. S., 20/30	O. D., secondary O. S., none
6	32241S	L	Intact (grossly)	O. D., 4 diopters O. S., 5 diopters	Not observed	O. D., none O. S., slight
Group 6B. Infiltrating (fig. 14)						
1	8556	L	Not observed	None	Not observed	None
2	7774	L	Defect in the right lower temporal quadrant; right hemichromatopsia (fig. 3a)	Bilateral, early	O. D., 10/30 O. S., 10/30	None
3	5983	L	O. D., temporal hemianopia (grossly) O. S., intact	None	Not observed	None
4	5978	L	Intact	Bilateral blurring	Not observed	None
5	8552	R	Intact (grossly)	O. D., 2 diopters O. S., 0.5 diopters	Not observed	None
6	37603S	R	Left homonymous hemianopia (fig. 3b)	O. D., 3 diopters O. S., 2 diopters	Good (grossly)	None

SUMMARY

The encapsulated posterior temporal lobe tumors gave inconstant and atypical defects in the visual fields; only in case 3 was there a quadrantic defect. In some cases papilledema was absent; in those in which it was present it was not particularly advanced; in some cases visual acuity was slightly depreciated or lost in one eye. Optic atrophy, primary or secondary, was occasionally found in the individual eye. In this group the differences in size of the pupil were at times similar to those found in the cases of infiltrating tumor; i. e., the larger pupil was on the side opposite the tumor. Impairment of convergence was frequent.

Characteristic defects in the visual fields were found most frequently in the infiltrating posterior temporal group. Unfortunately, the lesion was in some cases so advanced as to prevent perimetric studies. The lesion in other instances was of such character as not to interrupt the visual fibers, and hence no field defect was found. In the majority of instances, however, homonymous quadrantic or hemianopic defects led to the correct localization. In this group a certain constancy was found in the relation of inequality of pupils to the site of the lesion. Left-sided lesions were associated with larger pupils on the right, and right-sided lesions with larger pupils on the left. Impaired convergence, impaired conjugate movements of the eyes and paresis of upward gaze were frequent findings. Highly interesting is the fact that the preoperative period for the entire group was exceedingly short. Perhaps the early recognition and localization of the lesion with the aid of studies of the fields, leading to early operative procedure, was responsible for this.

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	R, good L, poor	Intact	(P), R>L (F), right	Convergence weakness on left	6 months	History of failing vision on left 6 months; on right, 1 month
R>L	Good	Intact	(P), R>L (F), right	Convergence weakness; weakness of upward gaze on left	6½ months	Patient aphasic, drowsy, uncooperative
R>L	Good	Intact	(P), L>R (F), right	None	1½ years	Death 4 months after craniotomy
L>R	R, good L, poor	Paresis of all branches of left 3d nerve (4th and 6th nerves intact)	(P), R>L ptosis of left lid (F), right	Left exophthalmos with downward displacement of left eye; vertical diplopia on upward gaze	6 years	Patient improved 8 months after craniotomy
R>L	R, fixed L, good	Intact	(P), R>L (F), right	Slight divergent squint with right eye rotated outward in midfixation	2 years	History of transient amaurosis on right 6 months; diminution of vision on left 4 months
Equal	Good	Slight palsy of left external rectus	Not observed	None	4 months	History of blurred vision 3 months; patient improved 7 weeks after operation
R>L	Good	Right external rectus palsy	(P), R>L (F), right	None	8 weeks	Patient uncooperative
R>L	Good	Intact	(P), R>L (F), right	None	7 weeks	History of diminution of vision 5 weeks
Equal	R, poor L, good	Intact	(P), equal	None	9 weeks	
Equal	Good	Intact	(P), equal	Impairment of conjugate deviation to right	3 months	
L>R	Fixed	Intact	(P), L>R (F), left	Impaired convergence bilaterally	9 weeks	
L>R	Good	Intact	(P), equal (F), none	Impaired convergence on left; slight impairment of upward gaze	4 weeks	History of diminution of vision and transitory diplopia 4 weeks; two operations within 5 months; death 7 months after last operation

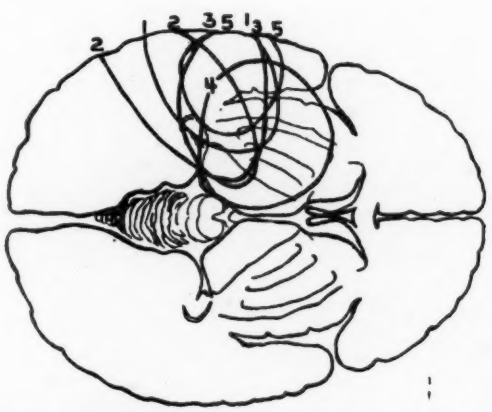

Fig. 14.—Location of posterior temporal infiltrating tumors (group 6B, table 7).

TABLE 8.—*Lob*

Group 7B.	Lobar infiltrating (fig. 14)						
1	5769	L	Not observed	O. D., 1.5 diopters O. S., 1.5 diopters	Not observed	None	Equ
2	3648	L	Not observed	O. D., 3 diopters O. S., 3 diopters	Not observed	None	R>I
3	5307	R	Not observed	Bilateral	Not observed	None	Not serv
4	3885	R	Not observed	Bilateral blurring	Poor	None	Equ
5	48498S	R	Defect in left upper quadrant (fig. 3c)	O. D., 5 diopters O. S., 6 diopters	Poor, R>L	None	L>I
6	41775S (X399)	R	Left homonymous hemianopia (fig. 3d)	O. D., 1.5 diopters O. S., 1.5 diopters	O. D., 10/30 O. S., 10/70	None	L>I

SUMMARY OF TABLE 8

In the cases of temporal lobar infiltrating tumor, because of the extent of the neoplastic processes which caused marked depreciation of the patients' intellect, failures to obtain reliable visual fields were frequent. Whenever obtained, significant defects in the fields were found. Papilledema was variable in degree; optic atrophy was absent; the pupillary changes were similar to those found in the posterior temporal group.

Equal	Good	Not observed	Not observed	Not observed	8 weeks	Patient died in coma before operation
R>L	Poor	Not observed	Not observed	Marked exophthalmos	4 weeks	Patient never recovered from stupor
Not observed	Fixed	Not observed	Not observed	Not observed	3 weeks	Patient died on admission
Equal	Poor	Right external ophthalmoplegia, complete; slight palsy of left external rectus	(P), L>R; bilateral ptosis (F), left	None	13 weeks	Patient uncooperative and died before operation
L>R	Good	Bilateral external rectus palsy R>L	(P), L>R (F), left	Right internal squint	4 months	History of diminution of vision 6 weeks
L>R	R, good L, poor	Intact	(P), L>R (F), left	None	7 years	History of diminution of vision 4 months

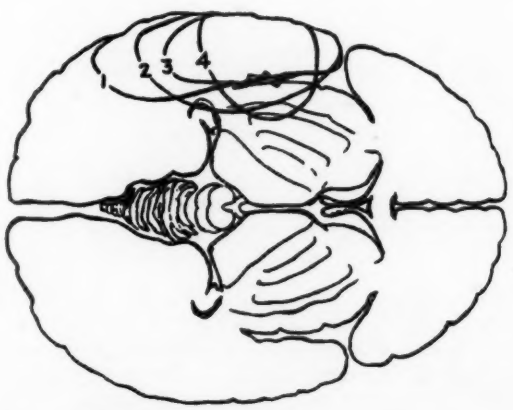

Fig. 15.—Location of temporal infiltrating lobar tumors (group 7B, table 8).

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 8A. Encapsulated (fig. 16)						
1	6519	L	Right homonymous hemianopia (fig. 3e)	None	Not observed	Bitemporal pallor (?) atrophy
2	6311	L	Not observed	Bilateral blurring	Not observed	None
3	8421	R	Intact	None	O. D., 15/20 O. S., 15/20	None
4	39557S	R	O. D., left homonymous hemianopia with contraction of temporal field O. S., no vision (fig. 3f)	O. D., 1.5 diopters O. S., 2 diopters	O. D., fingers at 1 ft. (33 cm.) O. S., amaurotic	Bilateral secondary
5	35052S (interlobar)	R	Defect in left upper quadrant (fig. 3g)	O. D., 2 diopters O. S., blurred	Poor (grossly)	None
Group 8B. Infiltrating (fig. 17)						
1	6390	L	Intact (grossly)	None	Good (grossly)	None
2	X360	L	Intact	None	Good (grossly)	None
3	48025S	L	Intact	None	O. D., 20/40 O. S., 20/40	None
4	P6567S	L	Intact (grossly)	Bilateral blurring	Not observed	None
5	X351	R	Left homonymous hemianopia (grossly)	Bilateral, early	Not observed	None
6	41966S	R	Intact	O. D., 1 diopter O. S., blurred	O. D., 10/15 O. S., 10/10	None

SUMMARY

Highly unusual and no less significant is the finding in the encapsulated parietal group of either hemianopic or quadrantic homonymous defects in the visual fields. Papilledema was less frequent and not high; visual acuity might be slightly diminished; secondary optic atrophy was rarely present. No pupillary changes were noted.

An analysis of the ocular manifestations in group 8 B reveals that the fields of vision were intact in the majority of cases. The record of hemianopia found in case 5 is of doubtful reliability. In 2 cases the inequality of the pupils was of the type seen in tumors of the temporal lobe. Papilledema was infrequent and, when found, was low. In all visual acuity was intact, and there was no optic atrophy.

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	Poor	Weakness of upward gaze on left	(P), R>L (F), right	Diplopia in all planes; occasional nystagmus on lateral gaze; conjugate deviation to right during epileptiform attacks—later, to left	5 years	History of diminution of vision 3 years; history of transient attacks of diplopia 1 year; patient aphasic, confused; status epilepticus; emergency craniotomy
Not observed	Not observed	Not observed	Not observed	Not observed	6 months	Patient died in coma a few hours after admission
Equal	Good	Intact	(P), equal (F), left	Occasional nystagmus on lateral gaze	6 months	Death 1 month after cervical laminectomy
Equal	R, good L, fixed	Intact	(P), L>R (F), left	Left eye does not converge	5 years	History of progressive diminution of vision
Equal	Poor	Intact	(P), L>R (F), left	None	2 years	Five operations in 9 months; patient unimproved 8 months after last operation
Equal	Good	Slight palsy of left external rectus	(P), L>R (F), right	Slight exophthalmos L>R; slight internal squint with right eye turned inward	3 months	Patient stuporous; emergency craniotomy
R>L	Poor (hippus)	Intact	(P), L>R (F), right	None	4 months	None
Equal	Good	Intact	(P), equal	None	4 months	History of blurred vision 1 week; patient improved 15 weeks after craniotomy
R>L	Good	Intact	Not observed	None	2 weeks	History of diplopia 1 day; patient improved after craniotomy
R>L	Poor	Not observed	(P), R>L (F), left	Right exophthalmos	2 weeks	Patient stuporous
Equal	Good	Intact	(P), R>L (F), right	None	6 weeks	Death 4 weeks after craniotomy

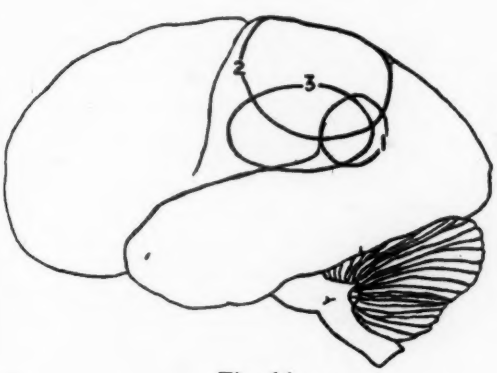

Fig. 16

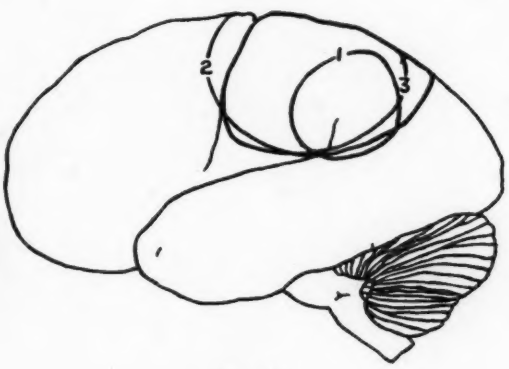

Fig. 17

Fig. 16.—Location of parietal encapsulated tumors (group 8A, table 9).

Fig. 17.—Location of parietal infiltrating tumors (group 8B, table 9).

TABLE 10.—O

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 9B. Infiltrating (fig. 18)						
1	6462	L	Not observed	Bilateral	Not observed	None
2	5842	L	Not observed	O. D., edema not measured O. S., 2 diopters	Not observed	None
3	7237	R	Left homonymous hemianopia (grossly)	O. D., 1.5 diopters O. S., 2 diopters	Not observed	None

SUMMARY

This group is too small to allow us to draw any conclusions. However, it is noteworthy that in case 3, in which cooperation on the part of the patient permitted studies of the visual fields, a homonymous hemianopic defect was obtained; also that the papilledema in all 3 cases was rather low. In none was there a history of visual hallucinations.

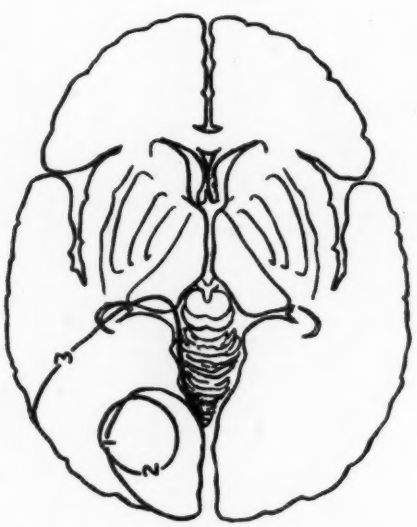

Fig. 18.—Location of occipital infiltrating tumors (group 9B, table 10).

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
R>L	Fixed	Not observed	Not observed	Divergent squint	1 month	History of diminution of vision 10 days; patient died in coma 24 hours after admission
Equal	Fixed	Not observed	(P), R>L (F), right	Conjugate deviation and nystagmus to left	3 months	History of blindness 3 days (?); Patient died in coma 6 days after admission
Equal	Good	Intact	(P), equal	Impaired convergence	3 months	Inability to open eyes, xanthopsia, diplopia 3 months

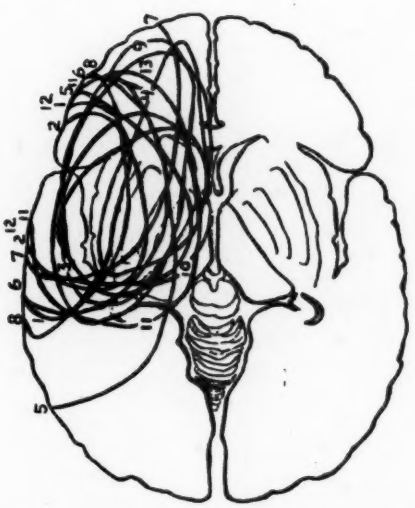

Fig. 19.—Location of temporofrontal infiltrating tumors (group 10B, table 11).

TABLE 11.—*Temp*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 10A. Encapsulated						
1	7910	L	O. D., intact O. S., no vision	None	O. D., 20/50 O. S., perception of light	O. D., none O. S., primary
2	33225S	R	Not observed	O. D., none O. S., early	O. D., amaurotic O. S., good	O. D., primary O. S., none
Group 10B. Infiltrating (fig. 19)						
1	8835	L	Not observed	Bilateral blurring	Not observed	None
2	8178	L	Not observed	None	Not observed	None
3	6763	L	Not observed	O. D., 2 diopters O. S., 3 diopters	Good (grossly)	None
4	6426	L	Not observed	O. D., 0.5 diopter O. S., 1.5 diopters	Not observed	None
5	3639	L	Not observed	Bilateral, old choked disks	Not observed	Bilateral secondary
6	X304	L	Right homonymous hemianopia (grossly)	Bilateral (not measured)	Good (grossly)	None
7	8575	R	Left homonymous hemianopia (grossly)	O. D., 2.5 diopters O. S., superimposed edema	Not observed	O. D., none O. S., primary
8	8261	R	Left homonymous hemianopia (quadrantic) (fig. 3h)	O. D., 3 diopters O. S., 2 diopters	Poor (grossly)	None
9	7891	R	Left homonymous hemianopia (quadrantic) (fig. 3i)	O. D., 7 diopters O. S., 5 diopters	O. D., 10/15 O. S., 10/20	None
10	7171	R	Not observed	Bilateral, early, L>R	Not observed	None
11	6520	R	(fig. 3j)	Bilateral blurring	O. D., 20/40 O. S., 20/40	None
12	5910	R	Left homonymous hemianopia (grossly)	O. D., 5 diopters O. S., 5 diopters	Not observed	None
13	3468	R	Intact (grossly)	O. D., 3 diopters O. S., 3 diopters	Good (grossly)	None

SUMMARY

In group 10 B, because of the poor mental state of the patients, visual fields were often impossible to obtain. When the patient's condition was such as to permit obtaining reliable fields, homonymous, quadrantic or hemianopic defects were obtained. It is rather striking how variable was the degree of papilledema. The papilledema seemed to be more marked on the side of the tumor. Optic atrophy was rare. Convergence, conjugate deviation of the eyes and upward gaze were frequently impaired. The pupillary differences were inconstant.

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Not observed	R, good L, poor	Left external rectus palsy	(P), R>L (F), left; left ptosis	Left exophthalmos 3 years; failure of convergence on left; impaired left upward and inward gaze	9 years	History of impaired vision 5 years with amaurosis 3 years on the left
R>L	R, fixed L, good	Intact	Not observed	Right divergent squint	8 years	History of right external squint 3 years; history of gradual loss of vision to amaurosis on right 3 years; Kennedy syndrome
Equal	Fixed	Not observed	Not observed	Conjugate deviation to left during attacks	1 year	History of diminution of vision on right for few months; patient died in coma few hours after admission
Equal	Good	Left external rectus palsy	(P), R>L (F), right	Conjugate deviation to left	3 days	Patient semistuporous, aphasic; death from pulmonary edema 3 days after admission
R>L	Poor	Bilateral external rectus palsy	(P), R>L (F), left	Impairment of convergence and upward gaze	1 year	Patient aphasic; mental changes
L>R	Fixed	Not observed	Not observed	External squint	4 weeks	Patient died in coma 5 hours after admission
Equal	Good	Intact	Not observed	None	Unknown	Patient stuporous, uncooperative; death 3 days after admission
L>R	Fixed	Not observed	(P), R>L (F), right	Conjugate deviation to right and upward during attack	6 weeks	
R>L	Poor	.....	(P), L>R (F), left	Paresis of convergence; occasional lateral nystagmus	3 weeks	
R>L	Fixed	Bilateral external rectus palsy	(P), L>R (F), left	Skew deviation; paralysis of convergence; slight limitation of upward gaze	4 years	History of blurred vision and diplopia 3 weeks
Equal	Good	Left external rectus palsy	(P), equal	Left eye lags in convergence; occasional left lateral nystagmus	3 months	History of diplopia and left external squint 6 weeks; history of blurred vision for few days
Not observed	Poor	Not observed	Not observed	Not observed	2 weeks	Died in coma 2 days after admission
Equal	Good	Intact	(P), L>R (F), left	None	1 year	History of "eye strain" for few months
Not observed	Not observed	Intact	(P), L>R (F), left	None	3 months	History of diplopia 1 week
Not observed	Good	Intact	Not observed	Lateral nystagmus R>L	4 months	Visual hallucinations

TABLE 12.—*Temp*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 11B. Infiltrating (fig. 20)						
1	8815	L	Not observed	None	Not observed	None
2	8481	L	Intact (grossly)	O. D., none O. S., slight blurring	Not observed	None
3	8183	L	Defect in right upper quadrant with uniform contraction (fig. 4a)	O. D., 4 diopters O. S., 4 diopters	O. D., 20/20 O. S., 20/20	None
4	8067	L	Not observed	None	Not observed	None
5	6728	L	Defect in right upper quadrant with uniform contraction	O. D., 5 diopters O. S., 4 diopters	O. D., 20/100 O. S., 20/40	None
6	6334	L	(fig. 4b)	O. D., 3 diopters O. S., 4 diopters	O. D., 20/20 O. S., able to count fingers	None
7	6077	L	Intact	None	Not observed	None
8	4238	L	Intact (grossly)	O. D., 3 diopters O. S., 3 diopters	Poor (grossly)	None
9	44112S	L	Right homonymous hemianopia	Bilateral, early	O. D., 20/100 O. S., 20/100	None
10	28938S	L	Right homonymous hemianopia (fig. 4c)	O. D., 4 diopters O. S., 4 diopters	Not observed	None
11	P6541S	L	Slightly contracted left field (fig. 4d)	Bilateral early blurring	O. D., 15/40 O. S., 15/100	None
12	6344	R	Not observed	O. D., 1 diopter O. S., 2 diopters	Good (grossly)	Bilateral secondary
13	6003	R	Not observed	Bilateral	Not observed	Bilateral pallor (? atrophy)
14	46969S	R	Left homonymous hemianopia (grossly)	O. D., 3 diopters O. S., 2 diopters	O. D., 10/20 O. S., 10/20	None

SUMMARY

In this group homonymous hemianopic defects (quadrantic or complete) were found whenever good cooperation could be obtained. Impaired convergence was also a frequent finding. Inequality of pupils was frequent, the larger pupil often being on the side opposite the tumor.

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
R>L	Good	Intact	(P), R>L (F), right	None	4 weeks	Patient aphasic, uncooperative
R>L	Good	Intact	(P), equal	Impaired convergence	1 month	Patient uncooperative
Equal	Good (hippus)	Intact	(P), R>L (F), right	Nystagmus on lateral and upward gaze	6 months	
Equal	Good	Intact	(P), equal	None	1 month	Patient aphasic, uncooperative; history of visual disturbance 1 month
Equal	Poor	Intact	(P), R>L (F), right	Impaired convergence; slight weakness of upward gaze; occasional right lateral nystagmus	2 months	History of diplopia 6 weeks
L>R	R, good L, fixed	Left 3d nerve paralysis	(P), R>L (F), right; left ptosis complete	None	9 weeks	Old chorioretinitis on left; history of left ptosis; history of diplopia 10 days
Not observed	Not observed	Right external rectus palsy	(P), R>L (F), right	Congenital right internal squint	3 months	
Equal	Good	Intact	(P), equal	Impaired convergence on left; weakness of conjugate deviation to left; nystagmus on left lateral gaze	10 weeks	History of impaired vision 2 weeks
L>R	Good	Left internal rectus weakness	(P), equal	Convergence weakness on left	3 months	History of diminution of vision 3 weeks; two operations in 10 months; patient improved 1 year after last operation
R>L	Poor	Left superior rectus weakness; right external rectus palsy	Not observed	Slight internal squint	6 weeks	History of diminution of vision and diplopia 3 weeks; improved after operation
Equal	Good	Not observed	Not observed	Not observed	2½ months	Condition unchanged 7 weeks after operation
Equal	Good	Intact	(P), L>R (F), left	None	Unknown	Mental confusion; patient uncooperative
Equal	Fixed	Not observed	(P), equal	Not observed	1 month	Patient died in coma a few hours after admission
L>R	Good	Intact	(P), L>R (F), left	None	6 months	Patient unimproved after operation

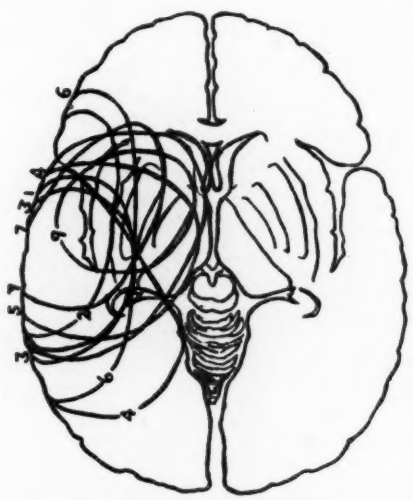

Fig. 20.—Location of temporoparietal infiltrating tumors (group 11B, table 12).

TABLE 13.—*Tempor*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 12B. Infiltrating (fig. 21)						
1	3977	L	Not observed	O. D., not observed O. S., none	Not observed	O. D., not observed O. S., none
2	41608S	L	Right homonymous hemianopia (grossly)	O. D., 2 diopters O. S., 4 diopters	Poor, right worse than left	None
3	8464	R	Not observed	Bilateral early	O. D., good O. S., poor	Bitemporal pallor (atrophy)
4	5609	R	Left homonymous hemianopia (grossly)	O. D., 2 diopters O. S., less, not measured	Poor (grossly)	None
5	5270	R	Left homonymous hemianopia	Bilateral early	Not observed	None
6	5151	R	Not observed	None	Not observed	None

SUMMARY

The visual fields, when obtained, showed homonymous hemianopic defects; the mental state of the patients often did not permit such studies. Papilledema was low or moderate and occasionally absent. Visual acuity was occasionally reduced, particularly when hemianopia was present. Optic atrophy was not present (except for the bitemporal pallor in case 3). The pupils as a rule were unequal; the larger one was consistently on the side opposite the location of the tumor. Upward gaze and convergence were frequently impaired; conjugate deviation of the eyes to the side of the lesion was present.

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Not observed	R, not observed L, good	Right internal rectus and left superior rectus weakness; left external rectus palsy	Not observed	(See last column)	10 weeks	Corneal opacity, anterior staphyloma, anterior synechia of right eye due to old injury; patient aphasic, semistuporous; acute otitis media; death 3 weeks after admission, before craniotomy
Equal	R, poor L, good	Bilateral external rectus palsy	(P), equal	Bilateral internal squint R>L	2 years	Formed visual hallucinations
L>R	R, poor L, fixed (hippus)	Bilateral internal and external rectus weakness	(P), equal	Slight impairment of upward gaze; bilateral exophthalmos; left internal squint; impaired convergence; conjugate deviation to right, incomplete	4 weeks	History of impaired vision 3 months; history of left internal squint and impaired convergence 2 weeks; patient in semistupor
L>R	Poor	Intact	(P), L>R (F), left	Impaired convergence	7 weeks	History of blurred vision 5 weeks
L>R	R, good L, poor	Bilateral external rectus palsy	(P), L>R (F), none	Occasional nystagmus on right lateral gaze; conjugate deviation to left during attacks	5 months	None
L>R	Good	Intact	Not observed	None	6 years	Mental deterioration; patient semistuporous; status epilepticus; death 4 days after admission

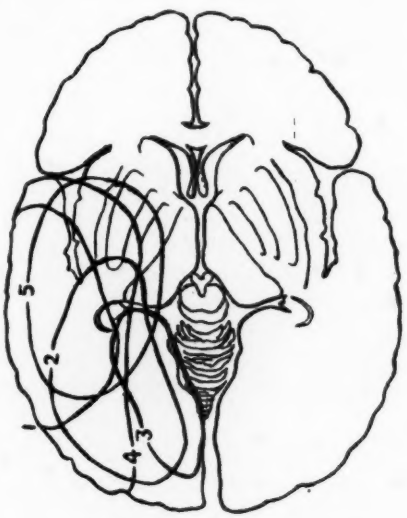

Fig. 21.—Location of temporo-occipital infiltrating tumors (group 12B, table 13).

TABLE 14.—*Occipi*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 14A. Encapsulated						
1	46686S	R	Left homonymous hemianopia (fig. 4e); visual defect on right side almost negligible	O. D., none O. S., 0.5 diopter	O. D., 20/20 O. S., 20/20	None
Group 14B. Infiltrating (fig. 22)						
1	7488	L	Right homonymous hemianopia (grossly)	O. D., 3 diopters O. S., 3 diopters	Not observed	None
2	5836	L	Right homonymous hemianopia (grossly)	O. D., 2 diopters O. S., 2 diopters	Not observed	None
3	5629	L	Right homonymous hemianopia (grossly)	O. D., blurred O. S., edema	Not observed	None
4	4578	L	Not observed	O. D., 4 diopters O. S., 4 diopters	O. D., good O. S., poor	None
5	8088	R	Left homonymous hemianopia (grossly)	O. D., 2 diopters O. S., 3 diopters	Poor	None
6	5900	R	Left homonymous hemianopia with contraction of fields (fig. 4f)	O. D., 4 diopters O. S., ?	Not observed	None

SUMMARY

In group 14 B the visual fields showed homonymous hemianopia uniformly; failure to obtain it was usually due to lack of cooperation. Papilledema was usually high. No reliable observations on visual acuity were obtained. Optic atrophy was not noted in any case. The pupils were unequal, the larger pupil almost always on the side opposite the tumor. Upward gaze was impaired in 2 cases.

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
R>L	Good	Slight palsy of left external rectus	(P), L>R (F), left	Slow nystagmoid jerks on right lateral gaze	3 years	History of "eye strain" 2 years, left homonymous hemianopia (grossly) 1 week; good result 10 months after operation
Not observed	Not observed	Not observed	(P), R>L (F), right	Not observed	2 months	History of diminution of vision 3 weeks
R>L	Good	Intact	(P), L>R (F), right right ptosis	Weakness in upward rotation of right eye; divergent squint	6 months	
R>L	R, fixed L, good	Right external rectus palsy	Not observed	None	3 years	
R>L	Poor	Slight weakness of left internal rectus; bilateral external rectus palsy	(P), R>L (F), left	Nystagmus on left lateral gaze	5 weeks	Patient 5 years old; uncooperative
Equal	R, fixed L, poor	Complete 3d nerve palsy on right; bilateral external rectus palsy	(P), L>R (F), left	None	2 months	
L>R	Poor	Congenital weakness of left internal rectus	(P), L>R (F), left	Impaired upward rotation; nystagmus on left lateral and vertical gaze; dissociation of conjugate movements to left	5 months	History of poor vision on left since birth; diminution of vision on right 3 months

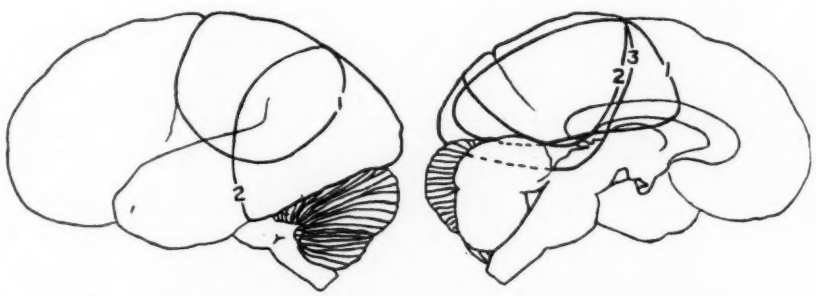

Fig. 22.—Location of occipitoparietal infiltrating tumors (group 14B, table 14).

TABLE 15.—*From*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 15A. Encapsulated						
1	45760S	L	Intact	Bilateral blurring	O. D., 15/15 O. S., 15/15	None
2	35757S	L	Intact (grossly)	Bilateral blurring	Good	None
3	27469S	L	Intact (grossly)	None	Good	None
4	4755	R	Left homonymous hemianopia (grossly)	O. D., 3 diopters O. S., 3 diopters	Good	None
Group 15B. Infiltrating (fig. 23)						
1	659S	L	Intact (grossly)	None	Good (grossly)	None
2	8893	R	Not observed	O. D., none O. S., not visualized	Good (grossly)	None
3	6733	R	Moderate contraction, especially left temporal field (fig. 4g)	O. D., early O. S., 2 diopters	O. D., 15/20 O. S., 8/20	None
4	5302	R	(fig. 4h)	Bilateral early	O. D., 20/30 O. S., 20/200	Bilateral secondary
5	4461	R	Intact (grossly)	O. D., 2 diopters O. S., 2 diopters	Not observed	None
6	41764S	R	Intact (grossly)	O. D., 1.5 diopters O. S., 1.5 diopters	Good (grossly)	None
7	34130S	R	Intact	Bilateral blurring	Good (grossly)	None

SUMMARY

Of interest in group 15 A is the contralateral enlargement of the pupil (2 cases) with hemianopic defects in only 1 case. Neither optic atrophy nor reduction of visual acuity was noted.

The 7 cases in group 15 B were characterized by the following disturbances: atypical contractions of the visual fields in 2 cases; low papilledema in all; diminished visual acuity in only 1 case; secondary optic atrophy in 1 case, and inequality of the pupils with a tendency for the larger pupil to be on the side opposite the lesion.

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
R>L	Good	Intact	(P), R>L (F), right	None	1 year	Improved 3 months after operation
Equal	Good	Slight palsy of right external rectus	(P), equal	None	4 years	Improved 2 years after operation
R>L	Good	Intact	Not observed	Left eye lags in convergence; lateral nystagmus and conjugate deviation to right during convulsive attacks	1 year	
Not observed	Poor	Bilateral external rectus palsy	(P), equal	Bilateral exophthalmos; occasional lateral nystagmus; impaired convergence	2 years	Two operations, second 7 years after first
Equal	Good	Intact	(P), equal (F), right	None	3 years	
R>L	Good	Intact	(P), equal	None	2 years	
L>R	Poor	Intact	(P), R>L (F), right	None	6 years	Poor cooperation
Equal	Good	Bilateral external rectus palsy L>R	(P), L>R (F), left	Convergent squint with eyes at rest; conjugate deviation to left during attacks	6 weeks	
Equal	Good	Intact	Not observed	None	2 years	History of diminution of vision 3 weeks
L>R	Poor	Right external rectus weakness	(P), equal	Left internal squint	3 months	Patient 3 years old; improved 7 months after operation
L>R	Good (hippus)	Intact	Not observed	None	5 years	Three operations in 2 years; death 2 years after last operation

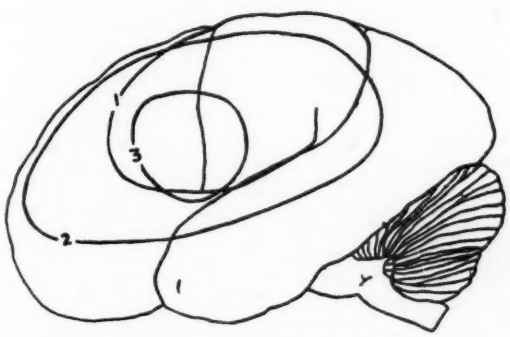

Fig. 23.—Location of frontoparietal infiltrating tumors (group 15B, table 15).

TABLE 16.—*Intraven*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 16						
1	6817	L	Intact	O. D., 2.5 diopters O. S., 2 diopters	O. D., 20/30 O. S., 20/20	None
2	4400	L	Not observed	None	Not observed	None
3	8066	R	Not observed	O. D., 1 diopter O. S., 1 diopter	Not observed	None
4	6241	R	Not observed	Bilateral	Not observed	None
5	4284	R	Intact (grossly)	O. D., 2.5 diopters O. S., 2.5 diopters	Not observed	None

TABLE 17.—*Tumors of the*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 17						
1	6453	L	Not observed	None	Not observed	None
2	6268	L	Not observed	Bilateral moderate	Not observed	None
3	5854	L	Not observed	Not observed	Not observed	Not observed
4	5828	R	Intact (grossly)	Bilateral	Not observed	None
5	3814	R	Not observed	O. D., 6 diopters O. S., 3 diopters	Not observed	None

SUMMARY OF TABLE 16

Intraventricular tumors (lateral ventricles) occur more frequently in children. This makes the study of the visual fields difficult and unreliable. In the 5 cases recorded here papilledema was usually moderate. The visual acuity could not be estimated accurately. Optic atrophy was not present. The pupils were usually equal.

ricular Tumors (Fig. 24)

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	Good	Intact	(P), equal	None	8½ months	History of blurred vision and diplopia 3 months
Equal	Good	Not observed	Not observed	Occasional nystagmus on lateral gaze; convergent squint	1 week	Infant; died 12 days after admission without operation
Equal	Fixed	Not observed	Not observed	Not observed	3 weeks	Moribund child, 3 years old; died in coma 7 hours after admission
Not observed	Poor	Not observed	Not observed	Eyes fixed, up and to the right	2 weeks	Infant; died 3 days after admission without operation
Not observed	R, poor L, good	Bilateral external rectus palsy	Not observed	Convergence weakness on right; limitation of upward rotation of left eye	8 weeks	History of photophobia 4 weeks; tuberosclerosis; patient aphasic, dioriented

Basal Ganglions (Fig. 25)

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	Fixed	Intact	(P), R>L (F), right	None	1½ years	Admitted in deep stupor; death 4 days after admission
Not observed	Not observed	Intact	(P), R>L (F), right	None	1 year	History of blurred vision 4 days; patient's cooperation poor because of choreiform movements
Equal	Fixed	Not observed	Not observed	Occasional nystagmus on lateral gaze	4 weeks	History of right internal squint and diplopia 4 weeks; died in coma 11 hours after admission
L>R	Good	Intact	Not observed	None	15 years	Tuberosclerosis
Equal	Good	Intact	(P), R>L left ptosis	Slight internal squint	2 months	Mental changes; patient uncooperative

SUMMARY OF TABLE 17

The visual disturbances in the 5 cases of tumor of the basal ganglions were as follows: The visual fields were not obtained, as the patients were usually too sick for such studies. Papilledema was variable in extent and occurrence. The visual acuity was not obtained. Optic atrophy was not noted in any case. The pupils showed no constancy in abnormal reactions.

Fig. 24.—Location of an interventricular tumor (group 16, table 16).

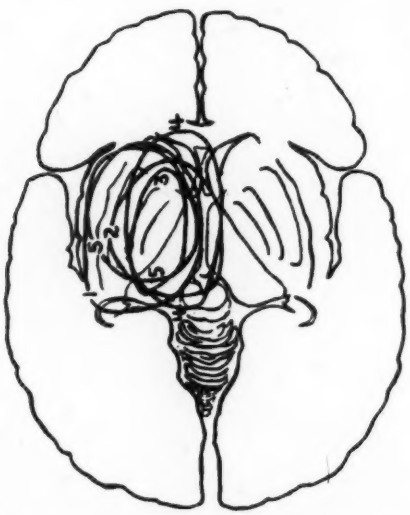

Fig. 25.—Location of tumors in the basal ganglions (group 17, table 17).

Fig. 26.—A suprasellar meningioma (group 18A, table 18).

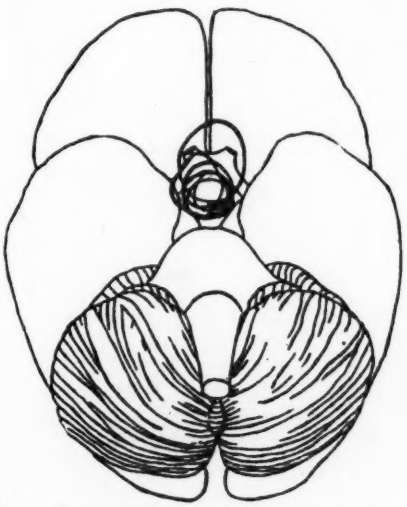

Fig. 27.—Location of the tumors of Rathke's pouch (group 18B, table 18).

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 18A. Suprasellar Meningioma (fig. 26)						
1	8109	—	O. D., temporal hemianopia O. S., no vision (fig. 4i)	None	O. D., 10/40 O. S., amaurotic	Bilateral primary L>R
Group 18B. Tumors of Rathke's Pouch (fig. 27)						
1	8859	—	Bitemporal hemianopia	Bilateral, slight, superimposed	O. D., 20/200 O. S., 6/200	Primary L>R
2	8719	—	Left temporal defect (grossly)	None	O. D., good O. S., poor	O. D., none O. S., primary incomplete
3	7104	—	Right homonymous hemianopia (grossly)	None	Not observed	O. D., primary ? O. S., secondary
4	4064	—	Contracted, especially temporally	O. D., 1 diopter O. S., 1 diopter	Not observed	Bilateral atrophy ?
5	3713	—	Not observed	None	Not observed	Atrophy ?; bitemporal pallor
6	X398	—	Slight uniform contraction (fig. 4j)	None	O. D., 10/70 O. S., 10/70	Atrophy ?; pale nerve heads
7	X313	—	Intact (grossly)	None	Good (grossly)	None
Group 18C. Pituitary Adenomas (fig. 28)						
1	8743	—	O. D., no vision O. S., temporal hemianopia (fig. 5a)	None	O. D., amaurosis O. S., 15/40	O. D., complete primary O. S., incomplete primary
2	8316	—	Bitemporal hemianopia (fig. 5b)	None	O. D., 20/50 O. S., 20/50	Bilateral incomplete primary
3	8092	—	O. D., no vision O. S., lower temporal quadrant defect (fig. 5c)	O. D., 2 diopters O. S., none	O. D., fingers not seen at 1 ft. (33 cm.) O. S., 10/100	Bilateral atrophy
4	7954	—	Not observed	None	Bilateral almost complete amaurosis	Bilateral, early primary
5	6274	—	Right homonymous hemianopia (fig. 5d)	Bilateral early, L>R	O. D., 15/15 O. S., perception of fingers at 3 ft. (91 cm.)	None
6	4023	—	Bitemporal hemianopia with moderate contraction of nasal fields (fig. 5e)	None	O. D., very poor O. S., poor	Right disk pale
7	47711S	—	Right temporal hemianopia (fig. 5f)	None	O. D., 15/30 O. S., 15/200	Bilateral primary L>R
8	47999S	—	O. D., small field preserved in temporal half O. S., upper temporal quadrant defect (fig. 5g)	None	O. D., amaurosis O. S., 20/20	O. D., advanced primary O. S., none

SUMMARY

In the 7 cases of tumor of Rathke's pouch the defects in the visual fields were variable; they included bitemporal hemianopia, homonymous hemianopia and unilateral temporal defects; in 1 case the fields were intact. Papilledema was rare, and if present, was superimposed on optic atrophy. Optic atrophy was primary and varied in degree. Inequality of the pupils was observed occasionally. The extrinsic ocular muscles often indicated involvement of the third nerve in some of its branches. Other changes were frequent (fig. 4j). Roentgen studies com-

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	R, good L, fixed	Bilateral internal rectus weakness L>R	(P), R>L (F), right	Occasional nystagmus on lateral gaze; convergence weakness on left	6 years	History of sudden onset of diminution of vision on left temporal side 8 months
L>R	R, fixed L, good	Intact	(P), L>R (F), left	Incomplete inward and upward rotation of right eye; right external squint	5 years	History of left divergent squint 1 year; erosion of sella turcica and posterior clinoids; suprasellar calcification
Equal	Good	Slight weakness of bilateral internal rectus	(P), R>L (F), left	External squint	3 months	Patient 3½ years old; erosion of sella turcica and posterior clinoids; suprasellar calcification
Equal	Poor	Right superior rectus weakness	(P), R>L (F), right bilateral ptosis	Slight vertical nystagmus	3 months	
Equal	Poor	Intact	Not observed	Conjugate deviation to right; slight nystagmus on lateral gaze	16 months	Normal sella turcica
L>R	Good	Right internal rectus weakness	(P), L>R (F), left	Right external squint	7 months	Patient 6 years old; enlarged sella turcica
Equal	Fixed	Bilateral 3d nerve palsy; right external rectus palsy	(P), R>L (F), right; bilateral ptosis	Right lateral nystagmus	2 years	History of squint and diplopia 1½ years; bilateral choroidal atrophy; erosion of sella turcica and posterior clinoids; suprasellar calcification
Equal	Good	Intact	(P), equal	None	6 years	Erosion of sella turcica and posterior clinoids; suprasellar calcification
R>L	R, fixed L, good	Intact	(P), R>L. (F), left	None	13 years	History of amaurosis on right 6 years; diminution of vision on left 1½ years
Equal	Good	Intact	(P), equal	None	15 years	History of impaired vision 8 years
R>L	R, poor (hippus) L, good	Intact	(P), equal	Impaired convergence	9 years	History of diminution of vision on right 9 years, on left 7 years
L>R	Fixed	Bilateral external rectus palsy R>L	(P), L>R (F), left	Weakness of upward gaze; right internal squint	4½ years	History of blindness 10 days
Equal	Good	Left external rectus paralysis	(P), R>L (F), none	Left exophthalmos; diplopia on left lateral gaze; left internal squint	10 years	History of impaired vision on left 10 years
L>R	Poor	Left superior and right internal rectus weakness	Not observed	Impaired convergence with right eye; left exophthalmos	6 weeks	History of diminution of vision 6 weeks; at necropsy residual tumor found in right frontal lobe
Equal	Good	Intact	(P), equal	None	4 years	History of diminution of vision, especially in lateral fields 4 years; vision not improved 6 months after operation
R>L	R, poor (hippus) L, good	Intact	(P), L>R (F), right ?	Tendency of right eye to turn outward on upward gaze	8 weeks	History of diminution of vision in temporal field of right eye 8 weeks

monly revealed calcific shadows. Frequently in the absence of shadows there was a large sella turcica with erosion of the clinoid processes.

In the 8 cases of pituitary adenoma typical bitemporal hemianopia was not the common picture; occasionally there was unilateral temporal hemianopia or a unilateral temporal quadrantic defect or homonymous hemianopia. Papilledema was rare, and when it occurred, it was superimposed on atrophy.

The visual acuity was reduced or lost. Optic atrophy was present in varying degree (no atrophy in 2 cases). Inequality of the pupils was frequent. The light reflex was often affected. The ocular movements were impaired.

Group 9 B. Occipital Infiltrating Tumors (3 cases; table 10 and fig. 18).—In only 1 of the 3 cases could the visual fields be studied; the examination revealed a homonymous hemianopic defect. There is reason to believe that similar results would have been obtained had the other patients been in condition to cooperate.

Group 10 A. Temporofrontal Encapsulated Tumors (2 cases; table 11).—This group was too small to yield convincing information; moreover, in both cases, because of primary optic atrophy, the studies of the visual fields were incomplete.

Group 10 B. Temporofrontal Infiltrating Tumors (13 cases; table 11 and figs. 3 *h-j* and 19).—Homonymous hemianopia was a frequent occurrence, with a relatively high incidence of the quadrantic form of the defect. In a large number of cases the lesion by reason of its loca-

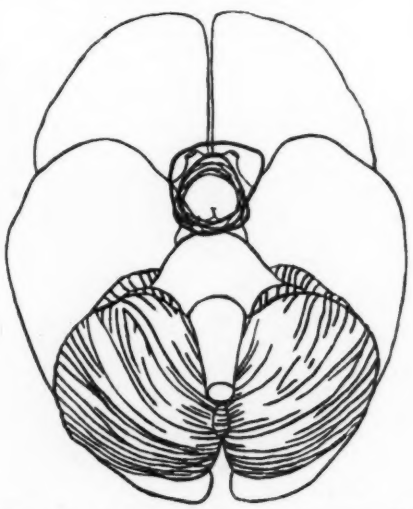

Fig. 28.—Location of pituitary adenoma (group 18C, table 18.)

tion and extent caused sufficient mental deterioration to defeat attempts at studies of the visual fields.

Group 11 B. Temporoparietal Infiltrating Tumors (14 cases; table 12 and figs. 4 *a-d* and 20).—Here also homonymous hemianopia with a high ratio of the quadrantic defect was a frequent occurrence; failure to obtain this result was usually due to the intellectual impairment of the patients.

Group 12 B. Temporo-Occipital Infiltrating Tumors (6 cases; table 13 and figs. 21 and 32).—In 3 of the 6 cases homonymous defects were present. In 2 of the patients the fields could not be studied.

Group 14 A. Occipitoparietal Encapsulated Tumors (1 case; table 14 and fig. 4 *e*).—Homonymous hemianopia was present.

Group 14 B. Occipitoparietal Infiltrating Tumors (6 cases; table 14 and figs. 4 *f* and 22).—In 5 of the 6 cases of this group homonymous

defects were found. Studies of the visual fields in the sixth case could not be carried out because of the youth of the patient.

Group 18 A. Suprasellar Meningioma (1 case; table 18 and figs. 4 *i* and 26).—The defects in the visual fields in this case were not unlike those found in other cases of suprasellar tumor.

Group 18 B. Rathke's Pouch (Suprasellar) Tumors (Craniopharyngioma) (7 cases; table 18 and figs. 4 *j* and 27).—Not unlike the subfrontal neoplasms, the tumors in this group, because of their proximity to the anterior (prechiasmal and chiasmal) segments of the optic path-

Fig. 29.—Infiltrating tumor of the third ventricle (group 19, table 19).

ways, yielded inconstant and irregular defects of the visual fields as follows: bitemporal hemianopia in 1 case, unilateral temporal defect in 1 case, homonymous hemianopia in 1 case, uniform contraction in 2 cases and intact fields in 2 cases.

This variability in the defects of the visual fields may in part be due to the frequent differences in the make-up and size of the respective tumors: Some of them were solid; some contained small cysts, while others were thin-walled cysts. Some remained limited to the interpeduncular space, while others expanded and implicated adjacent structures and the more posterior parts of the visual pathways.

TABLE 19.—*Tumors of th*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 19						
1	7337	—	O. D., no vision O. S., intact	O. D., none O. S., 2 diopters	O. D., perception of light O. S., 10/15	O. D., secondary O. S., none
2	7182	—	Not observed	None	Not observed	None
3	6293	—	Not observed	None	Not observed	None
4	6240	—	Intact (grossly)	None	Good	Bilateral primary
5	5469	—	Contracted (grossly)	Bilateral receding	Bilateral perception of light	Bilateral secondary
6	5368	—	Moderate uniform contraction	Bilateral early	O. D., 10/30 O. S., 10/10	None
7	4326	—	Intact	O. D., 4 diopters O. S., 3 diopters	O. D., 20/30 O. S., 20/70	None
8	3250	—	Not observed	O. D., 1.5 diopters O. S., early	Not observed	O. D., none O. S., incipient pallor

SUMMARY

In these 8 cases the visual fields were difficult to obtain because of a lack of cooperation on the part of the patients. Papilledema was low in 2 cases, moderate in 2 cases, high in 1 case, unilateral in 1 case and absent in 2 cases. The visual acuity, when obtained, was somewhat reduced. Optic atrophy occurred in a number of cases and was most often secondary (primary in 1 case). Exophthalmos was a frequent occurrence.

hird Ventricle (Figs. 29 and 30)

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	R, fixed L, poor	Intact	(P), equal	Bilateral exophthalmos; slight divergent squint	6 months	History of diminution of vision on right 6 months, on left 2 weeks
L>R	Good	Not observed	(P), L>R (F), left	Not observed	3 months	Mental changes; patient uncooperative
Equal	Good	Intact	Not observed	None	3 months	Patient drowsy and confused; genito-urinary infection; death before operation
Equal	Good (hippus)	Right external rectus palsy	(P), equal	Paresis of conjugate movements in all directions; rotatory nystagmus in right eye at rest and on lateral gaze; slight nystagmus in left eye	1½ years	Death 5 weeks after admission without operation
Equal	Poor	Intact	Not observed	Marked exophthalmos	6 months	History of diminution of vision 6 months; tuberosclerosis
Not observed	Not observed	Intact	(P), R>L (F), right	Bilateral exophthalmos; positive von Graefe sign	8 weeks	Uncooperative
Equal	Good	Intact	Not observed	Impaired convergence; slight exophthalmos; right lateral and vertical nystagmus	2 years	History of blurred vision 2 years; diplopia and left internal squint 1½ years
Not observed	Not observed	Not observed	Not observed	Not observed	6 months	History of diminution of vision on left 6 months; died in coma 24 hours after admission

Fig. 30.—Cystic encapsulated tumor of the third ventricle (group 19, table 19).

TABLE 20.—*Diffuse Hemispheri*

Case	Necropsy	Side	Visual Fields	Papilledema	Visual Acuity	Optic Atrophy
Group 20						
1	8712	L	Not observed	Bilateral blurring	Not observed	None
2	8653	L	Intact (grossly)	None	Not observed	None
3	8415	L	Not observed	None	Not observed	None
4	8133	L	Right homonymous hemianopia (grossly)	Bilateral early	Not observed	None
5	7472	L	Not observed	O. D., 3 diopters O. S., 2 diopters	Not observed	None
6	4303	L & R	Moderate uniform contraction (fig. 5h)	O. D., 6 diopters O. S., 6 diopters	Reduced to counting of fingers at 1 ft. (33 cm.) bilaterally	None
7	G5	L & R	Intact	O. D., 2 diopters O. S., 2 diopters	O. D., 20/30 O. S., 20/50	None

SUMMARY

In these 7 cases homonymous hemianopia was present when the temporo-occipital region was involved. Papilledema was noted in the majority of the cases, in varying degree. The visual acuity in 1 case was reduced; in others it was not recorded. Optic atrophy was present in 1 case.

Multiple Tumors (Fig. 31)

Pupils	Light Reflex	Extrinsic Ocular Muscles	Palpebral Fissures (P) Facial Palsy (F)	Additional Ocular Findings	Preoperative Period	Comment
Equal	Fixed	Intact	(P), equal	Eyes rolling about conjugately; slight exophthalmos; occasional left lateral nystagmus	4 months	Infant
Equal	Good	Intact	(P), equal	None	3 weeks	
Equal	Good	Left external rectus palsy (?)	Not observed	Conjugate deviation to left	2 months	Patient died in coma 2 days after admission
R>L	Poor	Intact	(P), equal	None	4 weeks	Patient died 2 weeks after admission, before operation
Equal	Good	Intact	(P), L>R (F), right	None	6 months	Mental changes; patient uncooperative
R>L	Poor	Bilateral external rectus palsy R>L	(P), L>R (F), left	Slight nystagmus on lateral gaze	3 weeks	History of diminution of vision and diplopia 3 weeks
Equal	Good	Intact	(P), R>L (F), right	None	3 months	Two operations in 10 months; death 1 year after last craniotomy

Fig. 31.—Diffuse hemispheric tumor (group 20, table 20).

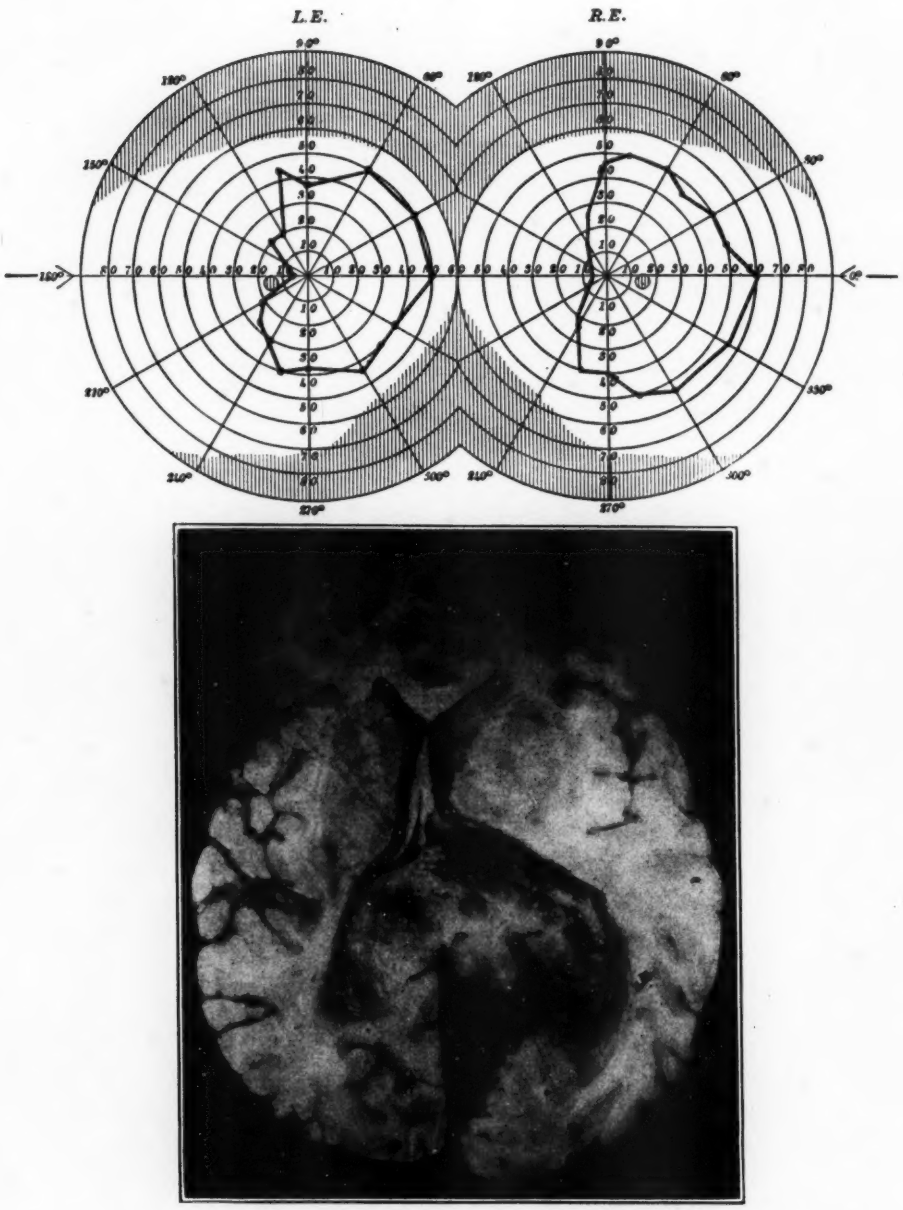

Fig. 32.—A tumor of the occipital lobe infiltrating the splenium of the corpus callosum. Above are the visual fields in the case.

Group 18 C. Pituitary Adenoma (8 cases; table 18 and figs. 5 *a-g* and 28).—Here also the typical bitemporal hemianopia was not the dominant visual defect. Again the close proximity of the tumor to the junction between the anterior and posterior segments of the optic pathways accounts for the variability in the defects of the visual fields, as shown in 6 of these cases: unilateral temporal defect in 3 cases, bitemporal hemianopia in 2 cases and homonymous hemianopia in 1 case.

It is evident that the classic bitemporal hemianopia is not common to all cases of pituitary adenoma. Unilateral temporal defects or even homonymous hemianopia are almost as frequent. Cushing [1] has already called attention to the fact that the bitemporal hemianopia in cases of pituitary tumor is but a stage in the development of the field distortions. This is readily recognized when field defects are detected early and the progress of field distortion is followed by successive studies repeated at short intervals. It then becomes apparent that the initial visual disturbances may at times take the character of a small unilateral temporal defect and often, instead of developing into a classic bitemporal hemianopia, may merge into a homonymous hemianopic defect (fig. 5 *d*).

Group 19. Third Ventricle Infiltrating Tumors (8 cases; table 19 and figs. 29 and 30).—No field defects were obtainable. This is rather unusual if it is recalled that the lesion here is very close to the optic chiasm.

Group 20. Diffuse Hemispheric Tumors (7 cases; table 20 and figs. 5 *h* and 31).—There was homonymous hemianopia in only 1 patient. The other patients were either too young or too sick to help in the study of the fields.

Comment.—Out of the 171 cases, there were 99 in which because of the anatomic relationship of the tumors to some of the segments of the optic pathways defects in the visual fields were to be expected. However, only 41 patients revealed disturbances in the fields which were of assistance in diagnosis. Of the other 58 patients 33 were unable to assist in carrying out these studies; 9 revealed intact fields; in these the examinations were gross only; 5 on careful perimetry showed normal fields; 9 showed contracted fields, and 2 patients were blind and hence could not be studied for field defects.

In going over the visual fields with the homonymous hemianopic or quadrantic defects it became apparent that the defect on the side opposite the lesion was almost always greater than that on the same side. Not only that, but it was possible to demonstrate by repeated

1. Cushing, H.: The Field Defects Produced by Temporal Lobe Lesions, Brain **44**:341, 1921; The Chiasmal Syndrome of Primary Optic Atrophy and Bitemporal Field Defects in Adults with a Normal Sella Turcica, Arch. Ophth. **3**:505 (May) 1930.

perimetry that in 1 case at least the early quadrantic defect occurred in the eye opposite the lesion while the homolateral field of vision was almost intact (fig. 5 *i*). An attempt to correlate these findings with the extent of the lesion found on postmortem examination did not yield reliable substantiating observations. This was due to the fact that in many of the cases operative intervention introduced factors such as hemorrhage or disorganization of the brain in the neighborhood of the tumor which probably did not exist at the time the fields were taken.

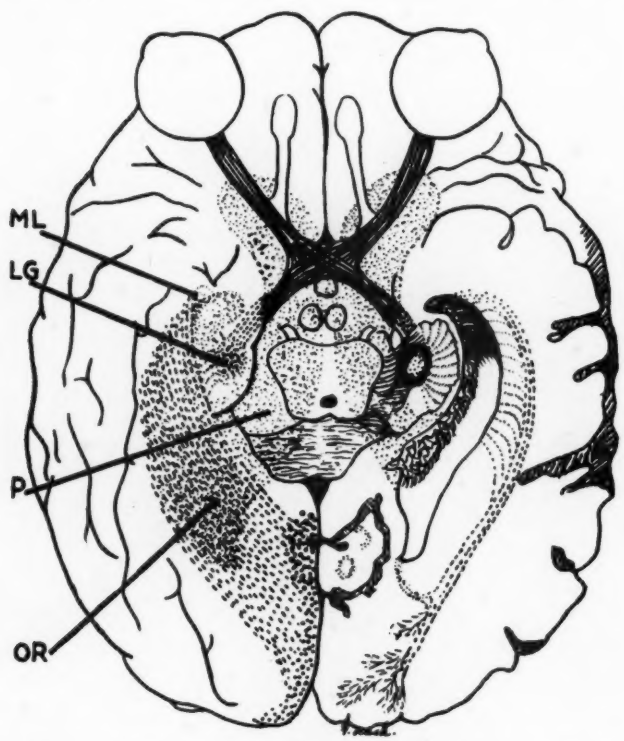

Fig. 33.—Course of the fibers in the optic radiations: *O R*, optic radiation; *M L*, Meyer's loop; *L G*, lateral geniculate body; *P*, pulvinar.

However, two explanations may be offered for this peculiarity in the defects of the visual fields: One is that a tumor advancing from within interrupts first the more median fibers in the loop of Meyer (fig. 33), which represent the contralateral nasal retina. But this is apparently not always true since tumors have been observed advancing from without which gave rise to the same quantitative differences in the fields (fig. 34). Hence it is necessary to consider the probability that the fibers in the optic radiation, in their course from the pulvinar and lateral geniculate body, rotate so that the nasal fibers are on the outside. In this manner a tumor affecting them first and to a larger extent will pro-

duce field defects greater on the same side as the tumor. It is planned at some later date to study the optic radiation in a group of such cases in order to substantiate the last suggestion or find another answer to the question. Of interest also is the fact that in a case in which the tumor extended from the occipital lobe into the splenium of the corpus callosum there was no loss of central vision (fig. 32). This is out of keeping with the belief that macular fibers pass through the corpus callosum to the opposite visual area, thus sparing central vision.

An attempt was made to correlate the precise seat of the tumor in the temporal lobe with the type of the associated homonymous field defect, but without illuminating results aside from the fact that a tumor

Fig. 34.—Large infiltrating tumor in the temporal lobe apparently advancing centrally.

situated in the anterior portion of the temporal lobe is not likely to produce any field defect of diagnostic import. A tumor, however, in the temporofrontal area, though the lesion is primarily in the anterior temporal region, may produce a homonymous quadrantic field defect. In such cases the tumor is apparently large enough to infringe on the most anterior fibers of Meyer's loop, or else this is caused by circulatory changes in tissue adjacent to the tumor. It is, indeed, disappointing to find that defects in the visual fields do not always allow a clearcut distinction between tumors in the posterior portion of the temporal lobe and those in the occipital lobe. The latter, particularly when overflowing into an adjacent lobe, may often be associated with field defects not unlike those seen with tumors of the posterior portion of the temporal lobe. It may nevertheless be true, as pointed out by Horrax and

Putnam,[2] that complete hemianopia is more characteristic of tumors that are strictly of the occipital lobe, while quadrantic defects in association with such tumors are the exception. It seems that without pneumoventriculography there is no reliable way of differentiating between a tumor of the posterior portion of the temporal lobe and one of the occipital lobe. A diagnostic lead may often be obtained when cerebellar signs appear alongside signs pointing to a supratentorial tumor in the temporo-occipital region, which suggest the probable location of the tumor as in the occipital lobe, most likely on its mesial surface and close to the midbrain.

B. In groups in which no defects of diagnostic value were expected the visual field findings were as follows:

Groups 2 A and 2 B. Prefrontal Encapsulated and Infiltrating Tumors (13 cases; table 3 and figs. 2 *c*, 7 and 8).—In 2 cases uniform moderate contraction of the fields was noted; in 6 cases the fields were intact; in the rest the fields could not be studied.

Groups 3 A and 3 B. Prefrontal Interlobar Encapsulated and Infiltrating Tumors (6 cases; table 4 and figs. 2 *d-e*, 9 and 10).—Slight contraction of the visual fields was present in 2 cases.

Groups 4 A, 4 A' and 4 B. Frontal Encapsulated and Infiltrating Tumors (16 cases; table 5 and figs. 2 *f-h*, 11 and 12).—Contraction of the visual fields was present in 3 cases.

Group 5 B. Temporal Anterior Infiltrating Tumors (5 cases; table 6 and fig. 13).—The visual fields were intact.

Group 8 A. Parietal Encapsulated Tumors (5 cases; table 9, figs. 3 *e-g* and 16).—Unexpected findings here were hemianopic defects in 2 cases and a quadrantic defect in 1 case. It is significant that tumors similarly located but infiltrating in character did not cause such defects.

Group 8 B. Parietal Infiltrating Tumors (6 cases; table 9 and fig. 17).—Here, in contrast to the findings in the cases of encapsulated parietal tumor, homonymous hemianopia was noted in a single case—on gross examination only and with the patient in a mental state not conducive to reliable observations.

Group 15 A. Frontoparietal Encapsulated Tumors (4 cases; table 15).—In only 1 case was a homonymous defect found preoperatively. The location and the extent of the neoplasm were discovered only at operation. Here, as in the cases of tumors of the parietal lobe (group 8 *A*), the encapsulated character of the neoplasm may be considered as a probable explanation of this unusual finding.

Group 15 B. Frontoparietal Infiltrating Tumors (7 cases; table 15 and figs. 4 *g-h* and 23).—Moderate contraction was found in 1 case.

2. Horrax, G., and Putnam, T. J.: Distortions of the Visual Fields in Cases of Brain Tumor, Brain **55**:499, 1932.

Comment.—Among the 171 cases there were 62 in which no disturbances in the visual fields were anticipated. In 3 of these cases homonymous defects were found. In these 3 instances the tumors were of the encapsulated variety. Slight or moderate uniform contraction was found in 8 cases. In all others the fields were intact.

C. In two other groups in which no prediction could be made as to the probable effect of the tumors on the visual fields studies of the fields yielded the following results:

Group 16. Intraventricular (Lateral Ventricles) Tumors (5 cases; table 16 and fig. 24).—No studies of the visual fields could be made because of the early age of the patients.

Group 17. Tumors of Basal Ganglions (5 cases; table 14 and fig. 25).—No defects of the visual fields could be found. It is quite probable that because of the seat of the lesion field defects would not have been obtained in any case even with fully cooperating patients.

PAPILLEDEMA

There is a strong inclination to believe that the farther posterior the location of a tumor the more likely is papilledema to be present, and the more pronounced is it bound to be. It is thought that with lesions in more posterior situations there is greater likelihood of obstruction to the drainage of ventricular fluid, and hence greater opportunity for the formation of internal hydrocephalus. Hydrocephalus is regarded by many as the most competent factor in the production of papilledema. This view, however, is contraindicated by the frequent observation that in communicating internal hydrocephalus there is very often no papilledema, and by the observations recorded in this paper. In our material advanced papilledema was often found with tumors of the frontal lobes which contributed little to the formation of hydrocephalus, while with lesions of the ventricular or temporal lobes commonly productive of hydrocephalus not infrequently the papilledema was often low or quite negligible. Hence some other factor besides the internal hydrocephalus must be invoked in explanation of the mechanics of the formation of papilledema. However, it should be said at this point that our observations apply only to supratentorial lesions and do not hold for tumors in the posterior fossa.

OPTIC ATROPHY

Optic atrophy, primary or secondary, is limited to a few groups. Primary atrophy is the more frequent and is usually found whenever the tumor is in direct relationship to the anterior segments, the prechiasmal or the chiasmal, of the optic pathways. The distribution of primary optic atrophy in our material is shown in table 21. It is obvious that primary optic atrophy is found in groups in which the

tumor is situated near the optic chiasm. The secondary condition is obviously not frequent and is perhaps a little more common when the lesion is infiltrative or is in the midline.

At this point it may be mentioned that the syndrome described by Kennedy [3] as characterizing expanding lesions of the frontal lobe, which under our classification would include prefrontal and subfrontal lesions as well, was noted in but 2 of 40 cases of tumors in these locations. Of significance, perhaps, is the fact that in both instances the tumor was a meningioma. In a case in which this combination of optic

TABLE 21.—*Primary Optic Atrophy*

Group	Cases	
	Unilateral	Bilateral
1A. Subfrontal encapsulated	1	2
4A. Frontal encapsulated	1* (contralateral†)	..
6A. Temporal posterior encapsulated	1 (homolateral†)	..
10A. Temporofrontal encapsulated	2 (homolateral)	..
10B. Temporofrontal infiltrating	1 (contralateral)	..
18A. Suprasellar meningioma	..	1
18B. Craniopharyngioma	2 + 2*	2
18C. Pituitary adenoma	1 + 1*	5
19. Third ventricle	..	1

* Questionable optic atrophy.
† Contralateral to or homolateral with the tumor.

TABLE 22.—*Secondary Optic Atrophy*

Group	Cases	
	Unilateral	Bilateral
1. Subfrontal	1	1
6A. Temporal posterior encapsulated	1 (homolateral)	..
10B. Temporofrontal infiltrating	..	1
11B. Temporoparietal infiltrating	..	2
15B. Frontoparietal infiltrating	..	1
18B. Craniopharyngioma	1	..
19. Third ventricle	1	1

atrophy on one side and papilledema on the other side was noted, the tumor was found on the side of the papilledema.

ACUITY OF VISION

Diminution of vision is generally found as an accompaniment of either primary or secondary optic atrophy and often in association with moderate or advanced papilledema. Of course, the objective manifestations, such as atrophy or papilledema, underlying the loss of vision serve as better localizing guides, and little reliance need be placed on the amaurosis itself.

3. Kennedy, F.: Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes, Am. J. M. Sc. **142**:355, 1911.

SCOTOMAS

An attempt was made to identify scotomas with tumors in certain locations. The survey of our material, however, revealed little of significance; color scotoma was noted in one eye of a patient with a subfrontal tumor, and an enlarged blindspot was found in one eye in a patient with a frontal encapsulated tumor. It is doubtful whether the tests were carried out with the precision and accuracy which would permit acceptance of the results as reliable.

PUPILLARY MANIFESTATIONS

Inequality of Pupils.—Of all the pupillary signs, the inequality of the size of the pupils is of particular interest. In all the groups, with few exceptions, and in a large number of cases the pupils were unequal. In many the inequality was unrelated to the site of the lesion, while in a large number of cases, particularly in those of anterior temporal, posterior temporal, temporal lobar, occipital, temporo-occipital, occipitoparietal, frontoparietal and diffuse hemispheric tumors, the larger pupil was very frequently contralateral to the site of the lesion; it was usually homolateral with the defect in the visual field. Highly interesting is the fact that in cases of intraventricular tumor and tumor of the basal ganglions and in a large number of cases of suprasellar tumor the pupils were frequently equal.

Light Reflex.—A poor reaction to light on one side was not infrequently found; it was not limited to any particular group. While in the larger percentage of cases the poorly contracting pupil was homolateral with the site of the tumor, there was no constancy in this phenomenon. In some instances vision was poorer in that eye. When the sluggish pupil was contralateral to the lesion it was usually associated with a homolateral hemianopic defect. One pupil fixed to light was noted in 7 cases. The majority of these instances were cases of sellar and suprasellar lesions. In such cases the direct involvement of the corresponding optic nerve by the tumor is to be held responsible for the condition. Bilateral fixation of the pupil was noted in 16 cases. The majority of these cases were those of sellar and suprasellar lesions, but other groups, particularly the various temporal infiltrating groups, contributed a fairly large number of instances with pupils fixed to light. The same holds true for 29 cases in which the pupils showed a poor bilateral light reflex. Here the representation is a much wider one. Practically each group represents a case in which there was bilaterally sluggish pupillary reaction. It is noteworthy that while impairment of vision due to optic atrophy or due to defects in the visual fields bears a relationship to the reduction of the light reflex, the papilledema seemed to bear no direct relationship to this manifestation.

PARESIS OR PARALYSIS OF CRANIAL NERVES SUPPLYING THE EXTRINSIC OCULAR MUSCLES

Abducens Nerve.—Of the extrinsic muscles of the eye, the rectus externus was most frequently involved. Only a few groups failed to contribute examples of such a condition (3 A, 3 B, 4 A, 4 A,[1] 4 B, 6 A, 8 A and 17). In 19 cases the paresis of the external rectus was bilateral; in 17 it was unilateral. In the latter cases, the external rectus was involved as often on the side of the lesion as on the side opposite the lesion. It is highly significant that the midline tumors, particularly those found in the interpeduncular space, were associated with palsies of the external rectus less frequently than were tumors in other locations. Tumors in the prefrontal and frontal region contributed a fairly large quota of palsies of the external rectus, both unilateral and bilateral. The unusual length of the sixth nerves, which is the cause given for the frequency of its involvement, may be considered for the present as the best explanation, for no lesion in the pons, aside from an occasional hemorrhage, could be found grossly to serve as a more convincing cause.

Oculomotor Nerve.—The next in frequency of involvement was the third nerve. Involvement of this nerve was more frequent in cases of prefrontal and interpeduncular lesions. It was also occasionally found in several of the temporal groups. The involvement of the oculomotor nerve was universally homolateral with the tumors. Here the explanation is found in the proximity of the tumor to the periaqueductal structure, the seat of the oculomotor nuclei.

Trochlear Nerve.—Involvement of the trochlear nerve is rather rare (in our material it was noted only in a case of complete ophthalmoplegia). The number of instances, however, might be much larger were the methods of detection of such paresis used with greater frequency and precision.

CONVERGENCE

Impairment of convergence is a frequent occurrence and was found in almost all of our groups with the exception of 2 A, 7, 14 A, 17 and 20. Groups 18 A, 18 B and 18 C are not included in this discussion for obvious reasons. It may be said that apparently impairment of convergence has no localizing value. The frequency with which it is found speaks for inaccuracy of observation. The examiner may be fully aware of the need of greater cooperation by the patient, whose concentration is essential for reliable findings. Impaired acuity of vision is another factor which contributes to the large number of such defects.

CONJUGATE OCULAR MOVEMENTS

In 6 cases there was irritative contralateral conjugate deviation of the eyes (conjugate spasm). In all of these cases this was associated

with jacksonian epileptiform attacks. In 7 cases the deviation was to the same side as the lesion and was unassociated with jacksonian attacks (conjugate paralysis). In 4 cases conjugate movement was impaired, and in 3 of these the conjugate movement was restricted to the direction of the lesion. In only 2 cases was the lesion in the frontal lobe; in the others it was fairly remote from the second frontal convolution, the seat of the center for conjugate movements of the eyes.

PARESIS OF UPWARD GAZE

Impairment of upward gaze was found in 13 cases. In all these the lesions were posterior to the boundary of the frontal lobe. Here, as with the palsies of the abducens and pupillary changes, the cause is to be found in the remote effects of the tumor on the structures in the quadrigeminate plate.

NYSTAGMUS

No true nystagmus was seen in association with any of the supratentorial lesions, but nystagmoid twitchings and inconstant fine oscillations were found in all the groups. No significance can be assigned to their absence or presence. Such disturbances were noted in 27 cases without being restricted to any particular group.

DIPLOPIA

A history of transient diplopia was obtained in 6 cases. In only 1 case was it present during observation.

EXOPHTHALMOS

Exophthalmos was noted in 12 cases. It was found, as would be expected, in the presence of subfrontal, prefrontal, frontal, temporal lobar, frontoparietal and third ventricle tumors. Unilateral exophthalmos was found in 3 cases and was homolateral with the lesion.

PALPEBRAL FISSURES

Inequality of the palpebral fissures was not a common finding. When found, it was usually in association with facial paralysis or paresis, which was usually part of a hemiplegic picture and contralateral to the lesion. Occasionally the wider palpebral fissure was homolateral with the lesion. Then it was a relative phenomenon because of ptosis on the opposite side.

COMMENT ON DISTURBANCES OF INTRINSIC AND EXTRINSIC OCULAR MUSCLES

To explain the frequent occurrence of involvement of the cranial nerves supplying the intrinsic and extrinsic muscles of the eyes as

well as of some of the associated movements of the eyes a systematic search for visible lesions in the brain stem was undertaken. The midbrain and pons were sectioned whenever available and inspected first for alterations visible to the naked eye. With a few exceptions in which some hemorrhages were noted, the pons was free from gross changes which could be held responsible for the paresis or paralysis of the abducens or the disturbances in conjugate movements of the eyes. On the other hand, in almost every case in which the third nerve was implicated, if no gross lesion such as a tumor was found in the interpeduncular space, some gross alterations were found in structures

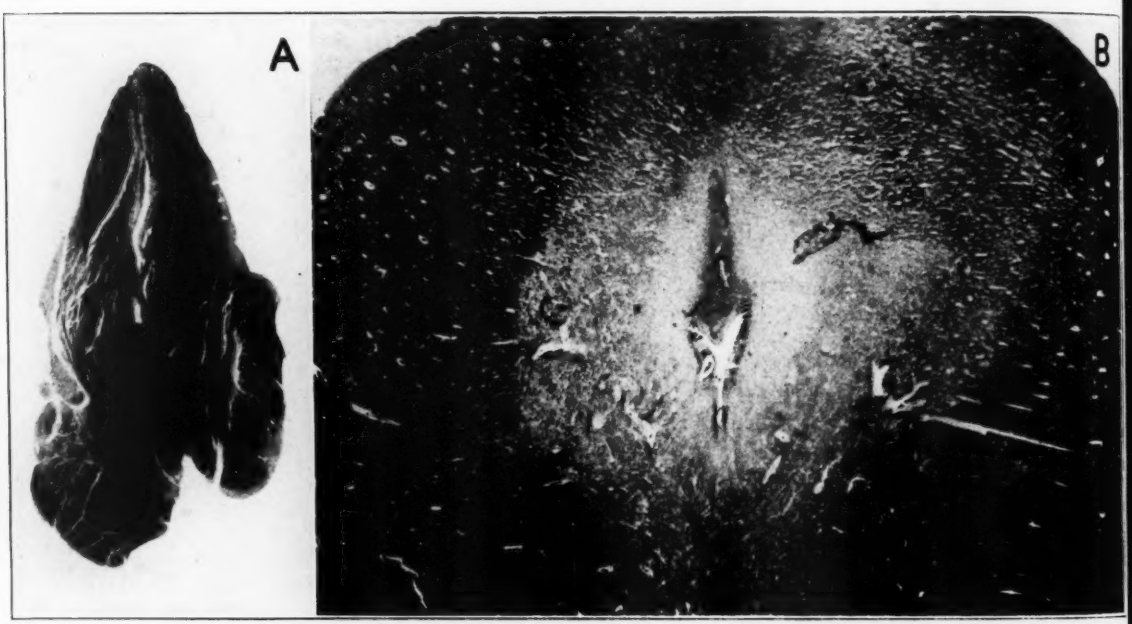

Fig. 35.—*A*, photomicrograph of a cross-section of the midbrain at the level of the anterior quadrigeminate bodies, illustrating the dislocation and distortion of the periaqueductal structures. *B*, photomicrograph of a cross-section of the midbrain at the level of the anterior quadrigeminate bodies, illustrating the exceedingly small aqueduct; × 7½.

neighboring on the aqueduct. There was gross displacement, distortion (fig. 35 *A*) or disorganization of such structures due to the proximity of the neoplasm. This was particularly true when the tumor was in the posterior temporal or the occipital region. In 3 instances the periaqueductal alterations were associated with an exceedingly (inherently) small aqueductal aperture (fig. 35 *B*).

Microscopic studies of such areas disclosed unquestionable pathologic changes in the radius of the oculomotor nerve nuclei in instances in which there was clinical evidence of their involvement. The changes were in

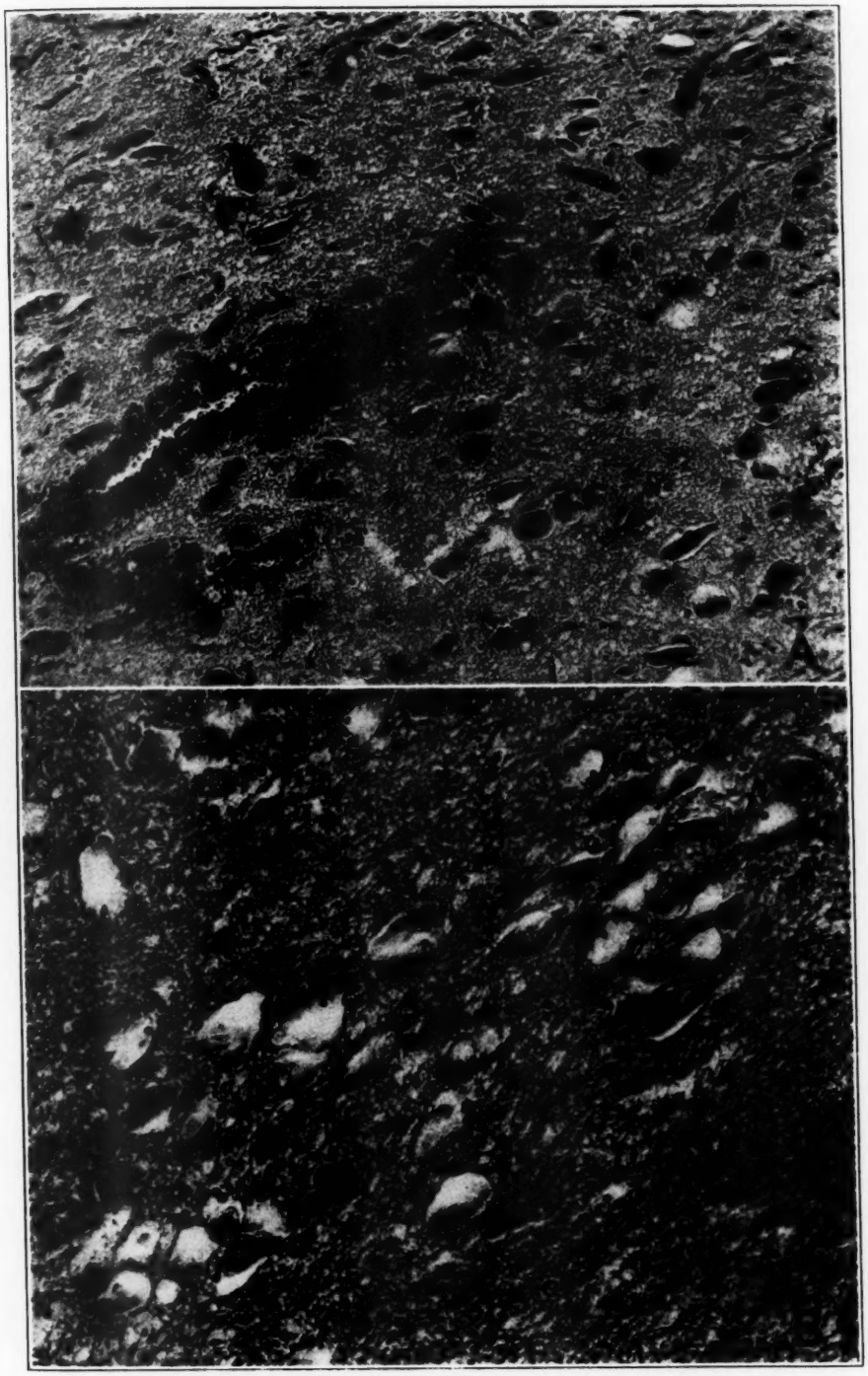

Fig. 36.—*A*, photomicrograph of a cross-section of the midbrain at the level of the anterior quadrigeminate bodies, illustrating conglomerate hemorrhages in the region of the oculomotor nuclei. *B*, photomicrograph of a cross-section of the midbrain at the level of the anterior quadrigeminate bodies, illustrating degenerative changes in the cells of the oculomotor nuclei.

the nature of extensive conglomerate petechial hemorrhages (fig. 36 *A*), perineural and perivascular edema and degenerative changes in the nerve cells in and about the oculomotor nuclei (fig. 36 *B*).

Significant is the fact that sections taken from the same areas in cases in which the oculomotor nerves were free from involvement displayed no pathologic alterations in the midbrain.

Thus it may be said that whenever the oculomotor nerve is involved in either its extrinsic or its intrinsic components or both the presence of a periaqueductal lesion may be predicted if the expanding lesion is not in the interpeduncular space. The involvement of the oculomotor nerve, of course, cannot be regarded as the result of increased intracranial tension, for in cases in which the intracranial tension is equally high, but in which the tumor is remote from the midbrain, these ocular findings are infrequent. Thus, it is more likely that the ocular manifestations are due to the direct effect of the tumor on the quadrigeminal area.

The frequent occurrence of impaired upward gaze in association with tumors neighboring on the anterior portion of the midbrain, which is considered by many to be the seat of a center controlling this mechanism, may also be explained by the conditions encountered in the periaqueductal region.

HALLUCINATIONS

In the material presented by Horrax and Putnam [2] visual hallucinations occurred in 15 per cent of the cases of tumor of the occipital lobe. In our material the number of tumors that were strictly of the occipital lobe is too small to permit any deductions in support of their observations. Nevertheless, it is quite appropriate for us to record that in none of our cases of tumor of the occipital or temporal lobe was there a history of visual hallucinations before or after operation.

PREOPERATIVE PERIOD [4]

The preoperative period, as we have already defined it, is the length of time elapsing between the appearance of the early subjective complaints which usher in the clinical course and the making of a diagnosis and taking of surgical steps. The mean preoperative period in our cases was seventeen weeks for the entire group with a longer period of seventy-three weeks for the encapsulated and a shorter one of thirteen weeks for the infiltrating tumors.

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4. Dr. Kennedy, in his discussion of this paper when it was presented before the Section of Ophthalmology of the New York Academy of Medicine, expressed the view that the low number of instances in which satisfactory studies of the visual fields could be made was due to the fact that such studies were undertaken late in the clinical course. That this apparently is not correct is shown by our survey of the preoperative period.

CONCLUSIONS

An analysis of the observations assembled in the tables reveals that as regards defects in the visual fields our material is divisible into three main categories:

I. In the first category are included tumors limited to the following groups: subfrontal (group 1), temporal posterior (group 6), temporal lobar (group 7), temporofrontal (group 10), temporoparietal (group 11), temporo-occipital (group 12), occipital (group 9), occipitoparietal (group 14) and interpeduncular (group 18).

The total number of tumors in these locations is 84. Forty-six of the tumors in these locations revealed characteristic defects in the visual fields (temporal and occipital groups) which were in accord with the accepted views as to the effects of an expanding lesion on any of the several critical segments of the optic pathways. In all these 46 cases the recognition of the visual disturbances was of decided assistance in making the preoperative diagnosis. In the remaining 38 cases reliable studies of the visual fields are not available because of the low mental state (semistupor, stupor) of the patients.

II. In the second category, out of a total of 22 cases (parietal [group 8] and frontoparietal [group 15]) there are 4 in which homonymous hemianopia was detected, but the lesions were ultimately found in regions outside the course of the optic pathways. Of interest is the fact that in 3 of the latter cases the tumor was encapsulated

III. In the third category are assembled tumors of the frontal lobe, and prefrontal tumors (groups 2, 3 and 4). Tumors in these locations are said to present a rather characteristic syndrome (homolateral optic atrophy and contralateral papilledema). In only 1 of the 40 cases was the Kennedy [3] syndrome found.

The three categories named account for 141 tumors of our material, leaving a balance of 30 tumors. Among the latter are 5 temporal anterior tumors (group 5), 13 intraventricular tumors (groups 16 and 19), 5 tumors of the basal ganglions (group 17) and 7 diffuse hemispheric tumors (group 20), in practically none of which diagnostic defects of the visual fields or other ocular changes of localizing significance were noted.

It is obvious that the number of tumors reported here in which perimetry was of diagnostic assistance is not large. This is a somewhat discouraging feature, particularly in view of the current belief that careful perimetry and other studies of the eyegrounds will serve as secure leads in the identification of the site and possibly the character of the expanding lesion. However, consideration must be given to the fact that attempts to obtain reliable visual fields were often frustrated by the poor cooperation of the patient or by the advanced diminution of

visual acuity caused directly or indirectly by the lesion. Of greater significance, however, is the fact that characteristic defects in the visual fields, when obtained at any period of the clinical course by reliable observers with the full cooperation of the patient, are of immense diagnostic service. It seems reasonable to assume that studies of the visual fields when carried out earlier in the clinical course may yield in a fair number of cases highly useful diagnostic leads for the localization of temporal, temporo-occipital and interpeduncular tumors; and that whenever defects in the visual fields are obtained under satisfactory conditions they are usually substantiated by operation or by the findings at necropsy.

It is, however, essential to bear in mind that there are several anatomic factors aside from the location of the tumor which frequently cause deviations from the characteristic visual disturbances. By virtue of its infiltrating character a tumor may spare the optic pathways as it invades the intercellular and interfibrous spaces, or it may encroach on the optic pathways in an irregular fashion, and thus cause great variability in the defects of the visual fields. Indirect pressure, particularly when caused by an encapsulated tumor, constitutes another factor which must be considered in an attempt to explain deviations from typical disturbances of the visual mechanism.

Among other observations which were recorded and discussed are: (*a*) the asymmetry of the defects in the visual fields in homonymous hemianopia, with the larger defect contralateral to the seat of the tumor; (*b*) the pupillary changes; (*c*) the involvement of the cranial nerves supplying the extrinsic muscles of the eyes, and (*d*) disturbance in the higher coordinating ocular mechanisms. They were commented on and correlated with the anatomic findings whenever possible. There are indications that such observations when carried out with the same precision and accuracy as the studies of the visual fields may render an exceedingly valuable service in the localization of tumors of the brain.

LATTICE TYPE OF CORNEAL DYSTROPHY

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Familial corneal degeneration or dystrophy was first described by Groenouw [1] in 1890 as the "nodular type of corneal opacities." Forty-three years later, in Breslau, Germany, at the joint annual meeting of the Elschnig and Bielschowsky clinics, I had the pleasure of appearing on the same program with Groenouw, who read a paper entitled "Nodular Type of Corneal Dystrophy Observed for Forty Years." At that time he stated that he was the only one who had personally examined all four generations in the same family.

Lattice-like dystrophy was first described by Haab [2] in 1898. An almost similar condition was described by Casper [3] as "lattice corneal opacity following injury," but Haab first stated that it was independent of ocular trauma. Fleischer [4] grouped together the nodular and lattice types as forms of familial corneal degeneration which appear varied although they have many characteristics in common.

In his original paper Haab spoke of alphabet keratitis, because the corneal opacities presented varying letter-shaped configurations. The condition began at puberty and was bilateral and accompanied by diminution of vision. His first cases were in members of the same family—a boy of 16 and his maternal aunt and uncle.

Dimmer [5] described the same entity almost simultaneously with Haab. He also observed three cases in the same family—those of two brothers and a sister. He added that recurrences of inflammation were seen in the first stages of the disease. This was possible because of the rubbing of the inner surface of the lid on the cornea.

From the Illinois Eye and Ear Infirmary.

1. Groenouw, A.: Knötchenförmige Hornhauttrübungen, Arch. f. Augenh. **21**:284, 1890.

2. Haab, O.: Die Buchstabenkeratitis, Beitr. z. Augenh. **10**:1, 1916; Die gitterige Keratitis, Ztschr. f. Augenh. **2**:235, 1899. Heine, L.: Die Krankheiten des Auges, part I, Berlin, S. Karger, 1921, p. 67.

3. Casper, L.: Gitterförmige Hornhauttrübung nach Augenverletzungen, Klin. Monatsbl. f. Augenh. **41**:289, 1903.

4. Fleischer, B.: Ueber familiäre Hornhautentartung, Arch. f. Augenh. **53**: 263, 1905.

5. Dimmer, F.: Ueber oberflächliche gitterige Hornhauttrübung, Ztschr. f. Augenh. **2**:353, 1899.

Pillat [6] saw the condition in a 20 year old patient in whose family the disease had occurred since its appearance in the great-grandfather. Jacqueau [7] observed the condition in a family of eight members, all of whom suffered from the dystrophy.

In contradistinction to Haab, Parsons [8] stated that there is little inflammatory reaction. He asserted that the opacities are limited to the center of the cornea and never invade the periphery.

The latest report in the literature is that of Frykholm,[9] who recorded an instance in which the condition was inherited through six generations. He described the clinical findings in thirteen members of the last three generations. This was the first time that the disease had been demonstrated in six generations. In his report cases were noted in twenty-one males and twenty-one females. The younger members showed lattice-like opacities, while the older members presented nodular opacities. He stated that this is further proof that the two types are related.

Fleischer expressed the belief that the same etiologic factors may be present in each type. Koby [10] stated that in many of the cases the condition appears to be due to chronic inflammation of the corneal nerves, to folds or ruptures in Bowman's membrane or to nodular swellings of the nerve endings. In Pillat's case thickening of the nerve endings was shown. Collins [11] expressed the belief that the lesion begins in the nerves and nerve endings and therefore is neurotrophic. Kraupa [12] and Löwenstein [13] agreed with this opinion. Working independently in the Elschnig clinic, they showed that these lesions represent a transition from the degeneration of the corneal nerves. On the other hand, Stanka,[14] also from the Elschnig clinic, used a

6. Pillat, A.: Ueber gitterige und andere Formen degenerativer Hornhauterkrankungen, Klin. Monatsbl. f. Augenh. **69**:682, 1922.

7. Jacqueau, L.: Une forme de kératite héréditaire et familiale, Clin. opht. **15**:434, 1809.

8. Parsons, J.: Diseases of the Eye, ed. 7, New York, The Macmillan Company, 1934, p. 237.

9. Frykholm, R.: Familiäre Hornhautentartung, vererbt durch 6 Generationen, klinisch nachgewiesen bei 13 Mitgliedern der 3 letzten, Klin. Monatsbl. f. Augenh. **94**:76, 1935.

10. Koby, F.: Slit Lamp Microscopy of the Living Eye, Philadelphia, P. Blakiston's Son & Co., 1925, p. 103.

11. Collins, T.: Hereditary Ocular Degenerations: Ophthalmic Abiotrophies, Tr. Internat. Cong. Ophth., 1922, p. 132.

12. Kraupa, E.: Die familiären degenerativen Hornhautveränderungen im System der sogenannten Dystrophien der Hornhaut, Klin. Monatsbl. f. Augenh. **70**:396, 1923.

13. Löwenstein, A.: Zur Klinik, Histologie und Therapie der gitterigen Hornhautdegeneration, Klin. Monatsbl. f. Augenh. **74**:357, 1929.

14. Stanka, R.: Familiäre gitterige Hornhautdegeneration, Klin. Monatsbl. f. Augenh. **74**:357, 1925.

10 per cent solution of brilliant cresyl to stain the cornea violet-blue and proved that the grill-work was not connected with the corneal nerves. Wehrli [15] suggested that the condition was an inactive form of corneal tuberculosis, but Schieck [16] stated that tuberculosis was probably an associated finding.

Pathologically, according to Haab, a transparent substance was deposited in Bowman's membrane. Exfoliated particles of cornea were examined chemically by Dimmer, who found a colloid or hyaline substance and triple phosphates. Greenwood [17] reported on a family of five afflicted members; he stated that a deposit of nonassimilable material is present. Goar [18] stated that subepithelial nodules occur, which consist of an amorphous substance staining yellow with trinitrophenol and resembling hyalin.

Fuchs [19] studied this type of keratitis thoroughly. He trephined a cornea involved by this process and described two changes which he stated were primary. A subepithelial secretion of a stratified substance is present, which takes an acid stain. There is also a basophilic nuclear mass in the cornea itself. Either condition or both occur, although the basophilic substance is always present. Formation of fissures, overgrowth of epithelium and thinning and disturbance of Bowman's membrane as well as swelling and partial disappearance of the corneal lamellae follow this change. This nutritional disturbance or dystrophy can be recognized by the secretion of a substance which does not dissolve in tissue fluid.

Paderstein [20] stated that a degenerative process takes place in the epithelium; the nuclei are destroyed, and the protoplasm is changed into a hyaline substance, which tends to accumulate on the surface of Bowman's membrane. Wirth [21] also noted a hyaline substance. Dr. Robert von der Heydt, who first observed the three cases which I shall present, has watched several cases over many years. In a personal communication he stated that in one case, in which he had made observa-

15. Wehrli, E.: Die knötchenförmige Hornhauttrübung (Groenouw) eine primäre, isolierte, chronische, tuberkulöse Erkrankung der vorderen Schichten der Cornea-Lupus Cornea, Ztschr. f. Augenh. **13**:322, 1910; Ein neuer Fall von knötchenförmige Keratitis, Klin. Monatsbl. f. Augenh. **47**:241, 1909.

16. Schieck, F.: Die Degenerationen der Cornea, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 4, p. 401.

17. Greenwood, A.: Lattice Keratitis, Tr. Am. Acad. Ophth. 1930, p. 248.

18. Goar, E.: Nodular Opacity of Cornea, Tr. Am. Ophth. Soc. **28**:91, 1930.

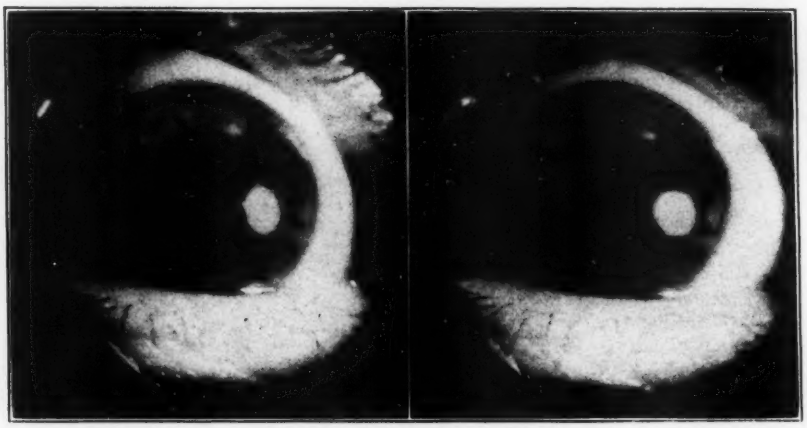
19. Fuchs, E.: Ueber knötchenförmige Hornhauttrübungen, Arch. f. Ophth. **89**:336, 1915.

20. Paderstein, R.: Bemerkungen zu Wehrli's Kritik meines Falles von knötchenförmiger Hornhautdegeneration, Klin. Monatsbl. f. Augenh. **47**:604, 1909.

21. Wirth, M.: Zur Histologie der knötchenförmigen Hornhauttrübung, Ztschr. f. Augenh. **58**:106, 1926.

tions with the slit lamp for the last eight years, there had been slow progression. The changes were in the stroma and consisted of a yellowish infiltration of the interlamellar cement substance. This case was previously described by him and Gradle in their stereophotographic atlas of the anterior segment of the eye.

Therapy in these conditions is symptomatic. An attempt is made to produce hyperemia and to increase metabolism by means of massage with ointment of yellow mercuric oxide, according to Schieck. Löwenstein recommended ethylmorphine hydrochloride and mercurial ointment (ethylmorphine hydrochloride, 0.3 Gm. and 33 per cent mercurial ointment and petrolatum, each 5 Gm.). Hot compresses may be of benefit. In addition, subcutaneous injections of iodine and subconjunctival injections of 1 cc. of a 4 per cent salt solution are given weekly.



Stereophotograph of the eyes of the patient in case 1, taken by Dr. von der Heydt.

The three cases which I shall report occurred in members of the same family, two sisters and a son of one of them. The son married his first cousin (the daughter of one of the afflicted sisters). The couple have three children who do not have the disease. The mother is also free from any signs of the condition.

REPORT OF CASES

CASE 1.—Mrs. A. A., an Italian aged 60, came to the clinic for glasses. She said that the vision in her left eye had been poor for the last five years, particularly during the last two months. Uncorrected vision in the right eye was 0.5; with the left eye counting fingers was performed at 10 feet (330.2 cm.). The pupils were equal and regular and reacted normally. Findings with the slit lamp were similar in the two corneas, except that the changes were much more marked in the left. Grossly the corneas appeared like parchment-paper, dry and some-

what lusterless. Irregular branching striations of a dull, dirty gray color were found with the slit lamp to be situated at the junction of the anterior and middle thirds of the stroma. The corneas did not stain; sensibility was normal. The tension in the right eye was 17 mm.; in the left eye, 21 mm. Medically and serologically, the results of the tests were negative. Chemical examination of the blood gave negative results. The lens, vitreous humor and fundus of the right eye were also normal. The fundus reflex was present in the left eye, but details were not distinctly seen.

CASE 2.—Mrs. J. C., aged 56, a sister of Mrs. A. A., was examined at the Illinois Eye and Ear Infirmary at the time the diagnosis in the case of her sister was made by Dr. von der Heydt. She complained of no visual difficulties. Uncorrected vision was limited to ability to count fingers at 3 feet (99.06 cm.) in the right eye and was 0.6 in the left. With a correction of +0.75 sph. ⊃ +0.5 cyl., axis 0, vision in the left eye was 1. Vision in the right eye could not be improved. The corneas were like those of her sister, the lesion in the right cornea in this case being more advanced. The results of medical examination and the Wassermann test as well as the blood chemistry and corneal sensibility were normal. The corneas did not stain. The tension in the right eye was 19 mm.; in the left, 21 mm.

CASE 3.—Mr. L. A., aged 37, a son of Mrs. A. A., was also examined when the diagnosis was made in the case of his mother. Uncorrected vision in each eye was 1. Grossly nothing abnormal could be seen in either cornea. With the slit lamp, however, incipient changes were definitely apparent. They appeared as fine, hairlike, glassy lines, irregular and branching. All possible etiologic factors were eliminated. The corneas did not stain. The sensibility was normal. The tension in the right eye was 21 mm.; in the left, 21 mm.

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LATE TRAUMATIC ROSETTE CATARACT

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It is obvious that trauma acting directly on the globe of the eye or on the parts nearby may give rise to a cataract. Although it is thus recognized that the development of an opacity of the lens may follow perforating injuries of the capsule of the lens or other objectively evident lesions of the anterior segment of the eye, there are cases in which it is difficult to assess what rôle, if any, has been played by trauma in the formation of a cataract. Thus, it may be impossible to elicit any history of injury, or a cataract may not have resulted in a loss of sight and so may have escaped subjective recognition, or the lesion of the anterior segment may not appear to be of a traumatic nature. In this connection arises the question whether or not the anterior cataract in the form of a rosette should be looked on as belonging to the traumatic variety—a question which opens a field for research which merits attention.

The studies of the last twelve years, that is, since Vogt[1] first described four cases in 1922, have cleared up many questions and have made a definite contribution to the knowledge of the morphogenesis of the lens, but the uncertainty which arises repeatedly, especially in cases involving compensation in which precise classification of the rosette cataract of Vogt is necessary, has not as yet been entirely dispersed. To elucidate these questions various factors must be taken into consideration.

The diagnosis of this condition is not always simple, for the location and sometimes the slight degree of the lenticular opacity facilitate its being overlooked during a hurried and superficial examination; again, the proper biomicroscopic technic is not universally known, and lastly, the condition may easily be confused with old opacities of the posterior cortex. Moreover, the situation has further been obscured by the various names which have been applied to the condition.

From the Institute of Ophthalmology of the Royal University of Bologna, Prof. Q. Di Marzio, Director.

1. Vogt, A.: Weitere Ergebnisse der Spaltlampenmikroskopie des vordern Bulbusabschnittes: IV. Abschnitt Cataracta traumatica und Cataracta complicata, Arch. f. Ophth. **109**:154, 1922; Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges, Part 2, Linse und Zonula, ed. 2, Berlin, Julius Springer, 1931.

The rosette cataract has been called the foliaform cataract (Rauh); it has been considered identical with the stellate form of Fuchs (Handmann), and it has also been described by Cattaneo under the name of radiating subcapsular cataract.

Without entering into the question of nomenclature, I have adopted Vogt's term, for it is to him that ophthalmologists owe the exact description of this particular clinical entity of the anterior cortex, whether or not it is associated with a lesion of the posterior cortex, and also because his term is that which corresponds best to the description by Schläpfer: *ein nach Form und Anordnung der Rosettenstrahlen charakteristisches Aussehen zeigt* (which as regards form and arrangement shows the characteristic appearance of the rosette rays).

The literature on the rosette cataract is very recent. After the first observations of Vogt (1922), in which he affirmed the traumatic genesis of the opacity, as well as its subcapsular origin, and in which he also brought out that the age of the cataract might be deduced as a result of the gradual displacement of the opacity toward the deeper strata of the cortex, no other work appeared until 1927. In that year Handmann [2] attempted to break down the rigid assertion of Vogt. Basing his opinion on eleven reported cases, in only two of which there was a complete rosette, he stated that he greatly doubted their traumatic origin, especially since there was no history of injury and no other objectively visible lesions were present in the anterior segment of the eye. As he did not have the facts necessary to clarify the origin of these rosette cataracts, he limited himself to describing them as belonging to one group.

Cases in which it has been impossible to ascertain the traumatic origin of the rosette cataract, either by history or by an objective examination, are not lacking in the literature. A few cases of Rauh [3] and one of Nordmann [4] are proof of this fact. Neither of these investigators, on the basis of these cases, attempted to assert that Handmann's view as to the nontraumatic nature of this type of cataract is true of all cases. Another contribution to the study of the rosette cataract was made by Schläpfer,[5] who in 1930 reported three cases.

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2. Handmann, M.: Ueber den Blatt-oder Rosettenförmigen vorderen Rindenstar, eine nichttraumatische stationäre Starform unbekannten Ursprunges, Klin. Monatsbl. f. Augenh. **78**:31, 1927.

3. Rauh: Blatt- oder rosettenförmige Stare, Klin. Monatsbl. f. Augenh. **84**: 766, 1930.

4. Nordmann, J.: A propos de certaines formes de cataracts en rosace, Arch. d'opht. **48**:392, 1931.

5. Schläpfer, H.: Weitere Beobachtungen über Kontusions-Spätrosette, mit Berücksichtigung rudimentärer Formen, Klin. Monatsbl. f. Augenh. **89**:346, 1932.

In 1932 Handmann [6] completed his first statistical study of twenty-eight cases of rosette cataracts, in nine of which the condition depended on a progressive trauma and in five of which it was nontraumatic and bilateral. After studying these cases Handmann admitted that the rosette cataract may be produced by trauma, but he expressed the belief that the fact that the opacity may occur bilaterally is strong evidence against the supposition that trauma is universally responsible.

Handmann's views were the object of severe criticism, especially on the part of Schläpfer, who accused him of having gathered together a number of instances in which the cataracts were heterogeneous as to form and pathogenesis and of having paid little attention to the histories.

Following this, Ascher [7] presented other cases of rosette cataract. In every one of his four patients, three men and one woman, the disorder was due to a contusion and involved predominantly the anterior cortex.

As the latest work, there appeared in 1934 a study by Cattaneo [8] on cataracts due to contusions, in which he considers the various forms of lesions of the lens in ocular trauma. He describes, however, cases of posterior subcortical cataracts, which correspond to either the rosette cataract or the radiate forms in the literature on the subject but which have absolutely no connection with those of the anterior cortex, which are later forms, much less frequent and of a different aspect. In not one of his observations was he able to note the characteristic picture which Vogt had called *traumatische Spätrosette*.

I have therefore deemed it opportune as well as interesting to present a few observations on the traumatic form of rosette cataract.

REPORT OF CASES

CASE 1.—*History.*—A. F., a farm-hand of Bologna, aged 57, did not recall having suffered from any serious general disorder. At the age of 10 years he was hit in the left eye by a stone, which resulted in external as well as internal lesions of the eye and for which he was treated at the clinic of the hospital at Castelbolognese. Three years prior to examination the patient fell from a wagon; this fall caused a lesion of the upper half of the left eye and also of the left knee (synovitis). He entered the clinic of the institute of ophthalmology complaining of a burning sensation, lacrimation and reddening of the left eye.

Ophthalmic Examination.—Vision in the left eye was perception of a moving hand at 25 cm.; the near point was indeterminable. Vision in the right eye was 10/10; with a +0.75 sph. it was 12/10. The near point was 12 cm., at which distance the third character was read. The pupils were anisocoric, the left more so

6. Handmann, M.: Weitere Mitteilungen über den Rosettenstar, Klin. Monatsbl. f. Augenh. **88**:601, 1932.

7. Ascher, K. W.: Die Kontusionsrosette der Linse, ihre Entstehung und ihre Bedeutung für die Unfallbegutachtung, Med. Klin. **29**:1045, 1933.

8. Cattaneo, D.: Osservazioni cliniche e ricerche sperimentali su le cataratte da contusione, Ann. di ottal. e clin. ocul. **62**:161, 1934.

than the right. The pupil of the left eye was irregular, with partial mydriasis; that of the right eye was normal.

Objective examination showed that the conjunctiva of the tarsus and fornix of the right eye was notably hyperemic. There was a catarrhal secretion at the internal and external angles. The fundus and media were normal.

Slit-lamp examination of the left eye gave the following results: The cornea presented incipient senile lesions (gerontoxon). Amorphous granules of pigment were irregularly scattered on Descemet's membrane. The anterior chamber was of ample size. The iris presented an atrophic stroma with a distinct depigmentation of the ectodermal layer, visible under reflected light. The pupillary border lacked pigment from 7 to 2 o'clock. The lens moved with the ocular movements,

Fig. 1 (case 1).—Microscopic appearance of opacities of the lens, showing floriform distribution.

and a posterior subluxation was present laterally. With mydriasis by atropine it was possible to see the equator internally, and through a semilunar fissure the vitreous humor was also visible.

Macroscopic examination of the left eye (objective 2, ocular 5×) showed that the lens was normal in form and somewhat reduced in thickness, with only the internal half visible, showing a bluish tinge along a narrow cortical zone at the equator; it was grayish and whitish in its depth. In the lens it was possible to distinguish opacities varying as to form, color and position, which were situated in the anterior cortex and on the anterior and posterior surfaces of the senile nucleus. Some of these opacities were pulverulent and some woolly, while others resembled either single or fused rose petals (fig. 1). These opacities involved various strata and delineated bands of disjunction or separation in the lenticular

substance. In front of the rosette opacity it was possible to distinguish five bands of separation, two of which were visible only under high magnification and in optical section while the other three were visible with ordinary means.

On further microscopic examination (objective 4, ocular 5 ×) it was impossible, despite all the optical means available, to detect any lesion of the capsule. On the capsule a stellate agglomeration of pigment granules was seen toward the central zone. Under the capsular epithelium it was possible to distinguish some transparent spherules, the smaller ones occupying the central zone, the larger ones the parapapillary zone.

It was possible to distinguish the first separation band at the equator only, since at the anterior pole it was confused with the capsule. Under it a thick fibrous stratum showing some lamellar dissociation presented, aside from the vacuoles just described, a few uniformly scattered pulverescent opacities. Internally, this first separation band limited a second thick stratum of transparent fibers which in optical section was not perfectly homogeneous but was made up of and combined with another thin stratum of transparent fibers in its posterior fourth. From 10 to 2 o'clock, toward the periphery, these strata contained a series of opacities which resembled open cotton fibers disposed radially. Toward 11 o'clock they resembled a large bubble with a clear transparent covering. Underneath these layers the cortex was divided into two other separation bands which were made up of beams containing some brownish and some grayish-white opacities with two other strata of transparent fibers interposed.

The opacities which made up the rosette cataract were seen astride the senile nucleus and involved more than one layer of fibers. They were made up of a close agglomeration of microscopic dots, disposed radially and separated by semitransparent zones. They resembled slightly the dotting of the "centralis pulverulenta." The margin of the opacity, in contrast with the central parts, was more marked, owing to the increase in volume of these opacities. The cataract was incomplete and practically limited to the internal superior quadrant.

The nucleus was normal in its entire thickness.

Other opacities were noted in the posterior half of the cortex throughout its entire thickness. At the level of the senile nucleus a wide, slightly stellate opacity, limited to a few layers of fibers, acted as an obstacle to the observation of the underlying opacities.

The vitreous humor was fibrous, with whitish accumulations along the fibers.

Case 2.—*History.*—W. S., a medical student of Bologna, aged 25, had had no preceding illness of note. At the age of 15, while playing with some schoolmates, he was hit in the left eye by a ball. He was taken immediately to a hospital for diseases of the eye, where he was given emergency treatment. No follow-up treatment was given. Four or five years later, while a university student, he noticed while doing microscopic work that the sight of his left eye was impaired. He visited a specialist, who prescribed glasses with plus lenses. Until the present no one has informed him of the presence of an anterior cataract.

Ophthalmic Examination.—Vision in the left eye was 6/10; with a +2.25 sph. it was 7/10. The near point was 20 cm., at which distance the second character was read. Vision in the right eye was 12/10; with a +1 sph. it was 12/10.

Slit-lamp examination showed that the right eye was normal. In the left eye, the cornea and iris were normal. The lens was of normal form, light bluish and not ectopic. In the lens it was possible to distinguish various types of opacities in the anterior and posterior cortex, which were so placed as to involve diverse layers of fibers. The anterior crystalloid, even with the use of greater magnifications, was of normal aspect. In the inferior half of the posterior portion, how-

ever, it was possible to appreciate an increased polychrome iridescence. Anteriorly, no subcapsular opacity was present. The first band of discontinuity was regular and was at the equator of the lens. As it proceeded toward the center of the lens it presented convexities and irregularities in its course, finally losing its identity by fusing with the capsule. Under it a thick layer of transparent fibers, in which were disseminated irregular, round and lumpy, whitish opacities, was observed. These opacities involved various layers and increased both in width and in number, proceeding from the center toward the equator and finally continuing in like manner in the same layers of the posterior cortex. In the anterior cortex it was possible to observe, in optical section, a grayish-brown band or ring which presented externally three opaque zones formed by a mass

Fig. 2 (case 2).—Microscopic appearance of opacities of the lens, showing rosette form.

of small grayish-white points disposed around the center of the lens in the form of a spindle. These were seen in various planes of fibers, assuming in optical section a seemingly ovoid form (fig. 2). They presented, therefore, a rounded surface anteriorly, raised above the normal plane of that layer, and a base which lost itself in the same stratum. At the margins this opacity was gradually attenuated in such a manner as not to cause the projection of the sutural zones (fig. 3).

In the vicinity of the equator the superior radial opacity continued with two other fan-shaped or rose petal-shaped opacities. Another opacity, placed laterally and on a higher plane, represented the anterior projection of the opacity of the posterior cortex.

Posteriorly, the inferior half of the cortex was transparent. The superior half was crossed by many clear serpiginous fissures and contained numerous

transparent spherules of various dimensions which were in turn limited by the opaque lenticular substance. All these features gave the tissue a spongelike appearance or one resembling a pumice stone.

The opacities of the posterior cortex disappeared at the equator. A few, however, were projected farther and continued on, diminishing in thickness in the anterior cortex and finally disappearing.

The nucleus of the lens showed distinct sutural lines and was normal in all respects.

The vitreous humor of the left eye presented a few copper-colored pigment granules.

Case 3.—*History.*—N. F., a farm-hand of Sesto Imolese, aged 19, stated that he had never suffered from any serious general disorder and that he had always been able to see well with both eyes. On May 20, 1932, while he was

Fig. 3 (case 2).—Microscopic appearance of opacity of the lens, showing attenuated margins.

watering oxen, one of the animals struck him in the left superciliary region with the point of a horn. An examination of the compensation report resulted in the disclosure of the fact that he had complained of an intense pain in the superciliary region, combined with a diminution of vision in the left eye.

Ophthalmic Examination.—Objectively, the patient presented a laceration 2 cm. long, which followed a transverse course along the internal portion of the superciliary region, involving the soft parts; a palpebro-orbital ecchymosis, and dialysis of the base of the iris in the internal superior quadrant, with consequent deformation of the pupil (Dr. Poggi).

A test of visual acuity in the left eye with the surprise system revealed a tendency to simulate vision of 5/10. Vision in the right eye was 10/10 without correction. The pupil of the right eye was central and reacted normally to light and in accommodation. That of the left eye was irregular and reacted to light and in accommodation. The fundus of each eye was normal.

Slit-lamp examination of the left eye showed an iridodialysis superiorly from 10:30 to 2 o'clock, which permitted exploration of the lenticular equator. The

pupillary aperture was consequently deformed and lowered. The deformity was maintained also under mydriasis by atropine.

Macroscopic examination of the left eye (objective 2, ocular 5×) showed that the lens was of normal form and in its normal position. It was not movable with ocular movements and was the color of greenish seawater. In the internal and superior half radial opacities, which originated at the lenticular center and proceeded downward toward 7 o'clock, internally toward 9 o'clock and upward toward 10:30 o'clock, were present. In an anterior stratum it was possible to observe a second zone occupied by a thin layer of opaque fibers which, from the sutural system, were distributed laterally from 10 to about 2 o'clock (fig. 4).

With a greater magnification (objective 4, ocular 10×) the capsule appeared normal in all respects. The first disjunction band was observed underneath the capsule, bounding a thick fibrous stratum which contained numerous scattered

Fig. 4 (case 3).—Microscopic appearance of opacities of the lens, showing formation with lateral distribution of fibers.

rounded grayish-white opacities, which in turn increased in size and number toward the lenticular equator. At the level of the second disjunction band a very thin stratum of opaque fibers made up of very light, radial opacities, and assuming a feather-like form was encountered.

More deeply, after passing a section of transparent fibers, a third separation band bounded the site of the rosette in front. Seen in optical section the rosette opacities were made up of very fine granules which increased in size toward the center and raised the line of demarcation of the separation band. In this manner various layers of fibers were involved. These opacities originated from the sutural system in the form of fine feather-like opacities. At the center they presented a more marked zone full of very fine opacities which caused the radial opacities to assume a lighter and more marked aspect. The remainder of the lens presented its normal sutural structure. The posterior cortex was normal in all respects.

Microscopic examination of the right eye showed nothing objectively visible aside from a few grayish opacities in the cortex, especially at the equator.

Case 4.—*History.*—P. G., a scholar of Oliveto, aged 13, declared that about a month before examination, while returning from school, he was struck in the right eye by a branch of a bush. At the time he complained of intense pain and a sudden loss of sight. After two or three days the pain ceased, but his sight showed no improvement, for which reason he was sent to this clinic by the Opera Nazionale Balilla (a Fascist children's organization) for examination.

Ophthalmic Examination.—With the right eye the patient was able to count fingers at 1.5 meters on the temporal side; the near point was 20 cm., at which dis-

Fig. 5 (case 4).—Microscopic appearance of opacities of the lens, showing feather-shaped character and radial distribution.

tance the twenty-eighth character was read. Vision in the left eye was 12/10 without correction; the near point was 20 cm., at which distance the first character was read.

Objective examination showed slight hyperemia of the tarsal conjunctiva and epibulbar and pericorneal hyperemia. The pupil of the right eye showed mydriasis, was irregular and reacted torpidly to light in its external half. The pupil of the left eye was rounded and central and reacted to light and in accommodation. The tension of the right eye was 41 mm. of mercury with 7.5 and 10 Gm. The tension of the left eye was 18 mm. of mercury with 5.5 and 7.5 Gm.

Slit-lamp examination of the right eye showed that the corneal epithelium was edematous. An irregular dispersion of pigment granules was present on Descemet's membrane. The aqueous humor showed accentuated tyndallization as well

as white and pigmented mobile suspensions. An unraveling of the pupillary border was noted in three distinct positions: at 8:30, at 10 and at 11 o'clock. The iris did not respond to light in the portion corresponding to the rupture of the sphincter. There was an accumulation of pigment granules on the stroma. Vitreous protruded through the border of the iris and entered the anterior chamber from above as well as from the external side. Suspended in this protrusion were various copper-colored granules.

The lens was subluxated and moved with the movements of the eye.

Microscopic examination showed that the lens of the right eye was of normal form, of a greenish seawater color and slightly luxated externally and posteriorly. It presented a great number of opacities which from the lenticular center proceeded, feather-shaped, toward the equator. They originated from the sutural system of the anterior cortex, assuming the form of many rays as well as evident radial direction (fig. 5).

Under greater magnification (objective 4, ocular 10 ×) no lesions of the anterior portion of the crystalloid were visible. Some of the opacities were seen to be situated underneath the anterior capsule and were represented by accumulations of whitish granules. Others, radially disposed, were seen along the course of the lenticular fibers. Between them numerous rounded vacuoles, uniformly scattered, were visible. In the internal half the opacities lost somewhat their radial disposition, becoming more coarse, and between them zones of normal transparency were visible. In the external half, especially in the external inferior quadrant, these opacities were distinctly limited by a more marked outline of pulverescent opacities which were slightly radial and which did not reach the extreme limits of the lens. Underneath these opacities the crystalline lens did not present any other lesion of note. The posterior cortex was normal in all respects.

In the vitreous humor white condensations and copper-colored granules were visible.

Nothing of note was presented by the left eye.

CASE 5.—*History.*—C. N., a housewife of Bologna, aged 53, declared that on Oct. 5, 1933, while in a butcher shop, she was struck in the left eye by a chip of bone. As a result of the trauma she suffered intense pain and a sudden loss of sight. She remained under the care of a local specialist until clinically cured. On May 3, 1934, she presented herself at this clinic for a control examination.

Ophthalmic Examination.—Vision in the left eye was 2/10; there was no improvement with correcting lenses. Vision in the right eye was 2/10; with a —7 sph. it was 10/10.

Slit-lamp examination of the left eye showed an irregular cicatrix which penetrated obliquely until it involved Descemet's membrane in the internal inferior quadrant of the cornea. The anterior chamber was of ample size. The stroma of the iris was regular but contained an accumulation of pigment. The pupil was irregular, ovoid and adherent to the anterior portion of the crystalloid by means of a whitish bridle which contained pigment granules. The crystalline lens was the color of greenish seawater, immobile and adherent by means of a capsular synechia to the posterior surface of the pupillary border. At the point of attachment of the synechia the capsular surface presented a hollowing out, around which might be observed a series of whitish punctate opacities which involved the upper layers of the anterior cortex. Posteriorly, they were continuous with a lamellar dissociation zone. In the anterior cortex, anterior to the anterior surface of the adult nucleus and in the midst of the cortical zone, the fibers which originated from the sutures presented a very light opalescence which under greater magnification seemed to be composed of a great number of pulverescent opacities radially dis-

posed. There was nothing of great note concerning the other strata of the embryonic nucleus.

In the right eye the outline of the senile nucleus was very evident on slit-lamp examination. In the cortex it was impossible to observe anything which might be likened to the changes in the left eye.

Case 6.—*History.*—L. P., a laborer of Zola Predosa, aged 55, stated that while working on the construction of a paper mill near Stuttgart, Germany, in October 1903, he was struck over the right superciliary arch by a piece of brick. He was taken to a hospital, where he was obliged to remain for fifty-one days. He was then discharged but with greatly reduced vision in the right eye. For

Fig. 6 (case 6).—Microscopic appearance of eye, showing rosette opacities.

a few years he has not been able to distinguish other than the motion of a hand before this eye. Recently he complained of diminution of the vision of the left eye.

Ophthalmic Examination.—Vision in the left eye was 5/6; there was no improvement with correcting lenses. With the right eye the patient could distinguish motions of a hand at 20 cm.

Slit-lamp examination of the right eye showed that the central part of the cornea was transparent. At the periphery, however, there were two arched zones where the epithelium was turbid. Underneath the epithelium numerous small yellowish spherules were observed, some of which seemed to be situated underneath Bowman's membrane. In the parenchyma the senile arc was not very marked. The anterior chamber was shallow superiorly but deep inferiorly. There was a slight dispersion of the light in the aqueous humor. Iridodonesis was noted. The stroma of the iris was somewhat vague in its outline. A posterior synechia extended throughout the superior quadrant from 10 to 2 o'clock. The lens was

subluxated superiorly and anteriorly. It was possible to observe a slightly turbid fibrillary structure immediately underneath the capsule. Numerous whitish punctiform opacities were observed disseminated in the anterior cortex. In the anterior strata of the adult nucleus five dense whitish foliaform opacities were observed. Under greater magnification these opacities were seen to be made up of an agglomeration of whitish points which followed the course as well as the peripheral projection of the lenticular fibers. They were perfectly delimited by transparent zones. The superior half of the lens could not be explored, owing to the adherence of the pupillary border. It was possible, on the basis of the general aspect of the opacities, to hold that the rosette was complete. The posterior cortical strata were more visible and presented only a light dissemination of whitish punctiform opacities.

The fundus could not be explored.

The tension was 24 mm. of mercury with 7.5 and 5.5 Gm.

COMMENT

The observations which may be made from the examination of patients 1, 2, 3, 4 and 6 are many and involve, aside from the relationship between trauma and opacity, (*a*) the site of the opacity, (*b*) the form, (*c*) the etiology, (*d*) the course and (*e*) the complications.

Vogt, in his first work on this type of traumatic cataract, stated that the initial opacities are manifested in the anterior cortex, immediately underneath the capsule. They are represented by a mass of small opacities which radiate peripherally from the sutural lines, making visible the anatomically known course of the lenticular fibers in their projection toward the posterior sutural system. In this manner figures of geometric type are formed, all of which are radially disposed.

Considering my figures, it is noted that a certain number of variations exist. From the typical rosette figure seen in cases 2, 3 and 6, despite the incompleteness or the association with other opacities, one passes to the floriform appearance noted in case 1 and finally to the typical initial form in case 4. There have been cases in which the sutures, owing to the fusion of the coarser opacities with them, were marked, as demonstrated in cases 3 and 4. There was a minor involvement of these sutures in cases 1 and 6, and finally there was complete independence of the opacities from the sutures in case 2. Aside from the accumulations of fine whitish pulverescent opacities, identical with those which go to make up embryonic central congenital cataracts such as seen in cases 1 and 2. I also observed, in cases 3 and 4, coarser opacities. Under observation with appropriate means, however, they corresponded to masses of pulverescent opacities, which were more evident the more they involved the various strata of fibers and which, disposed along the course of the fibers, produced the rosette figure.

It is clear that this variability must be dependent on three specific factors which may be listed as: the mode of action, the intensity of the

trauma and the time between the traumatic action and the observation of the eye.

These three factors cannot be divided, since they act together to give the clinical entity the more or less diverse aspect already noted. If this were not so, all other opacities which might be determinable in the course of time and which are nothing but variations of the complicated and presenile forms, would not be explicable. The trauma, acting on the bulb with a certain violence, frequently causes lesions of the anterior segment. Examples of this are: the iridodialysis in case 3, the unraveling in case 1, the rupture of the sphincter in case 4, the luxation of the lens in cases 1 and 6, the secondary glaucoma and the presence of the vitreous humor in the anterior chamber in case 4 and the posterior synechia in case 6. Therefore, there occurs a series of alterations in the lens which involve not only the anterior cortex but the posterior as well. It is precisely to this that one must attribute the cortical opacities, both anterior and posterior, which I have observed in practically all the cases. By their late appearance they demonstrate more and more the manner in which the distance of the rosette opacity from the anterior capsule may represent the age of the cataract.

The secondary opacities no longer have the aspect of posttraumatic rosette opacities but present themselves in the more common form of less coarse masses disposed in the upper strata. Through their disposition they demonstrate an element which is now beyond discussion, i. e., the growth of the lens through the juxtaposition of new fibers.

It is therefore possible to note, especially in case 1, that the primitive cortical zone was restricted and on a plane inferior to that which corresponds to the anterior surface of the senile nucleus. Over the rosette opacity, it was possible to note two strata of opaque fibers which alternated with transparent layers, bounding, therefore, as many secondary discontinuity bands. Other opacities, placed above the first, were visible in the cortex and corresponded to the various stages in the formation of the additional fibers.

The age of the cataract may, therefore, be ascertained with accuracy from the distance between the anterior disjunction band and the plane of the rosette opacity. I have not been able to make any exact measurements, but the case histories have been substantiated by the position of the opacities. The depth of the opacity was greatest in cases 1 and 6, in which the trauma dated back forty and thirty-three years, respectively; it was medium in case 2, in which the trauma dated back only ten years, and practically nil in case 4, in which the trauma dated back but one month. The two remaining cases gave intermediary values. These observations, therefore contribute to the study of the growth of the lens and knowledge of the etiology and evolution of this type of cataract. The question of the etiology of the clinical entity of the

rosette cataract was not discussed by Handmann in 1927. At first Vogt's thesis of traumatic origin was taken for granted without discussion.

After the time of Handmann, however, views changed, since other authors, Rauh and Nordmann, happened to find a few instances in which it was impossible to ascertain the traumatic origin in any way. Despite that fact, Handmann's idea did not meet with the success that his vigor in proposing it led one to believe it would. This was not so much because observers had felt, as he affirmed, the influence of Vogt—an influence which at the most might have affected Schöpfer,[9] since he is a disciple of the Zurich clinic—but because the traumatic factor is so evident.

Summary of Cases

Case	Patient	Sex	Age, Years	Site of Lesion	Time of Trauma	Site of Lesions in Lens	Other Lesions
1	A. F.	Male	51	Left eye; vision = movement of hand at 28 cm.	About 40 years before	Anteriorly at the senile nucleus; posteriorly throughout the cortex	Unraveling of the sphincter; atrophy of the iris; subluxation of the lens; vitreous in anterior chamber
2	W. S.	Male	25	Left eye; vision = 6/10	1924	Anterior cortex and superior half of posterior cortex	Nothing of note; copper-colored pigment granules in the vitreous humor
3	N. F.	Male	19	Left eye; vision = 5/10	5/20/32	Anterior cortex	Dialysis of the iris superiorly and internally
4	R. G.	Male	13	Left eye; vision = 1/10	4/20/34	Anterior cortex	Rupture of the sphincter; subluxation of the lens; vitreous in anterior chamber; secondary glaucoma
5	G. N.	Female	53	Left eye; vision = 1/10	5/10/33	Capsule and anterior cortex	Corneal cicatrix; posterior synechia
6	L. P.	Male	55	Right eye; vision = movements of hand at 20 cm.	1903	Capsule and anterior cortex	Large posterior synechia; subluxation of the lens; chronic iridocyclitis

If one glances over the table giving the summary of the cases, one is able to conclude that: men are most frequently affected; the young are most usually affected; usually only one eye is affected; the trauma was objectively indeterminable in only one case (case 3); complications involving the anterior segment of the eye were present in all the cases; this form of cataract may be considered as stationary. It is therefore the objective examination which makes one sustain the idea of the traumatic origin of the rosette and not the authority of Vogt.

Another question which merits particular attention is that regarding the course of the cataract. It is known that these opacities, which are

9. Schöpfer: Ueber die traumatische Spätrosette, Klin. Monatsbl. f. Augenh. **85**:276, 1930.

initially subcapsular, slowly shift from their original location to occupy, finally, the deep layers of the cortex. Among my six cases a few demonstrate this. For example, in case 4 the cataract was initial and subcortical, whereas in two other cases (1 and 2) the rosette cataract was complicated by other opacities, both anterior and posterior, the latter being more pronounced than the former.

The opacities observed in case 4 only a short time after the trauma have permitted me to confirm Vogt's theory on the subcapsular genesis of the traumatic rosette and to refute, along with Nordmann, that of Handmann. If one considers the other two cases, one may even reconstruct the evolution of the traumatic rosette. In both cases, aside from the rosette opacity of the anterior cortex, there existed also opacities in the posterior cortex which had all the aspects of being of the complicated or secondary form. If these opacities had been caused by the rupture of the capsule, the capsule in healing would have caused the formation of a circumscribed opacity, and the apposition of the fibers either would not have taken place or would have brought about the formation of circumscribed opacities in the cortex behind the nucleus. This did not happen, since the fibers of the rosette cataract were not seen in a more cortical plane of the remaining zone.

The juxtaposition of the fibers had taken place regularly. In case 2 a greater polychrome refraction of the inferior half of the posterior capsule and a total opacity of the superior half were noted. Underneath this capsular opacity the entire cortex of the lens was involved. The same may be said of case 1.

I have therefore observed a pathologic process of the lens which had involved at the same time the new formation of fibers, since the final opacities stopped exactly in the plane which constituted the anterior surface of the rosette. If this were not so, why did the trauma cause only one opacity so deeply situated?

I hold, therefore, that the rosette cataract should be placed among the stationary forms. The secondary opacities are observed when the trauma has caused a lasting unbalancing of the nutritive mechanism of the lens or when the ocular lesion has determined a chronic irritation of the posterior part of the uvea.

Many authors have mentioned stellate opacities of the posterior cortex. Both Vogt and Cattaneo place these opacities on the same plane as the rosette and have mentioned them as an involvement of the posterior cortical stratum. I have far too few illustrative elements to form a personal opinion in regard to this. I shall limit myself to saying that I have not seen the "bird feather" form described by Vogt and Cattaneo. I have seen the stellate form—a form which limits anteriorly the opacities of the posterior cortex—in only one case (case 1). The posterior opacities in case 2 did not show any resemblance to the common descrip-

tions of the posterior stellate forms. Because of this I believe that they are all late forms and in our cases dependent on the trauma, since it was this which caused the permanent lesions and the chronic irritation of the anterior segment of the ocular bulb.

CONCLUSIONS

The following conclusions are drawn:

The rosette cataract is a well differentiated form of cataract which is formed in the anterior cortex as the result of a trauma.

The rosette cataract must be considered as stationary.

The ultimate involvement of the lenticular fibers must be considered as related to the trauma only in the sense that this trauma was the cause of grave lesions of the anterior segment which brought about an unbalancing of the physiologic nutrition of the lens.

Therefore, my observations answer affirmatively the question: "Should the rosette cataract be considered a form of traumatic cataract?"

LAROCAINE, A NEW ANESTHETIC

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For the past two years the literature, more especially that in German, has brought forth the study and evaluation of new anesthetics for local and subcutaneous use. During the first part of 1932 the Hoffmann-LaRoche Company sent me a quantity of larocaine for trial. During the progress of my study, various authors, to be cited later, have published articles concerning this anesthetic. Therefore, this paper will be presented as a confirmation and substantiation of previously published findings, together with additions which will make the American reader acquainted with the product.

Larocaine, a synthetic product, is the *p*-aminobenzoyl ester of 2,2-dimethyl-3-diethylamino-1-propanol. It is supplied in the form of a hydrochloride, a white crystalline powder soluble in water up to 50 per cent. Chemically, it is akin to procaine hydrochloride and tutocaine. It is stated that the lethal dose for the mouse is 0.3 Gm. per kilogram of body weight by subcutaneous injection, compared with 0.1 Gm. of cocaine, while the lethal dose for the rabbit is 0.15 Gm., compared with 0.07 Gm. of cocaine. These statements have been corroborated in my experiments.

The initial pharmacology of larocaine was worked out by Fromherz.[1] The chemical formula was found to be $(C_2H_5)_2N.\ CH_2C\ (CH_3)_2.\ CH_2\ OOC.\ C_6\ H_4NH_2.\ HCl$. Fromherz evaluated the drug on the rabbit's cornea and the spinal cord of frogs and found it to be twice as efficient as cocaine in the former, and to have twice the power of procaine hydrochloride in the latter. It was much less toxic on the exposed frog's heart than cocaine. A sublethal dose was excreted in forty-five minutes and could be followed by a like dose at that time without any signs of an accumulated effect. Dietrichs [2] found larocaine to be more rapidly excreted than most of the other benzoyl esters. Epinephrine increased the length of the anesthesia produced by larocaine. In an excellent review and history of the pharmacology of local anesthetics, Hirschfelder and Bieter [3] summarized the knowledge of larocaine in its

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1. Fromherz, K.: Arch. f. exper. Path. u. Pharmakol. **158**:368, 1930.
2. Dietrichs, H.: Arch. f. exper. Path. u. Pharmakol. **161**:206, 1931.
3. Hirschfelder, A. D., and Bieter, R. N.: Physiol. Rev. **12**:190 (April) 1932.

relation to other anesthetics. In seeking a substitute for 10 per cent cocaine, Koch [4] rejected tutocaine, pseudococaine, alypin and nupercaine, but found a 5 per cent solution of larocaine satisfactory.

In the field of general surgery, the following authors have reported on larocaine. Schildbach [5] performed 350 operations of various kinds, using from a 0.5 to a 1 per cent solution, and he felt that the preanesthetic period was shorter with larocaine than with procaine hydrochloride. Volkmann [6] found larocaine efficacious in a 1 per cent solution, and he used solutions up to 10 per cent subcutaneously, which took the place of a 20 per cent solution of cocaine. Kemkes [7] concluded that larocaine fulfils the postulates of Braun,[8] in that it is easily sterilized, gives proper anesthesia and may be reinforced by epinephrine.

In the field of ophthalmology, three reports have been made. Weiss [9] used compressed tablets containing 0.003 Gm. of larocaine which could be handily inserted in the lower culdesac. The tablets are sterilized and are easily kept in this state. When the burning ceases after the tablet has been inserted, anesthesia is complete enough for all ordinary office procedures. Balcarek [10] has made larocaine the anesthetic of choice in all cases in which it is indicated in the practice of ophthalmology. For corneal and conjunctival application, one drop of a 1 : 1,000 dilution of epinephrine precedes the use of a 5 per cent solution of larocaine in physiologic solution of sodium chloride. Balcarek found no clouding of the cornea as with cocaine, and no hyperemia as with pseudococaine, phenacaine and *p*-n-butylamino-ethanol of dimethylamino-ethanol (pantocaine) even if the epinephrine is omitted. Even after many hours no enlarged pupils were encountered. In operations on the lids with the Gonin technic and in operations on muscles a 2 per cent solution gave excellent anesthesia, and the patient was comfortable. Ocular tension could be taken one minute after instillation of the drug, and foreign bodies that had been burned in were removed after two minutes.

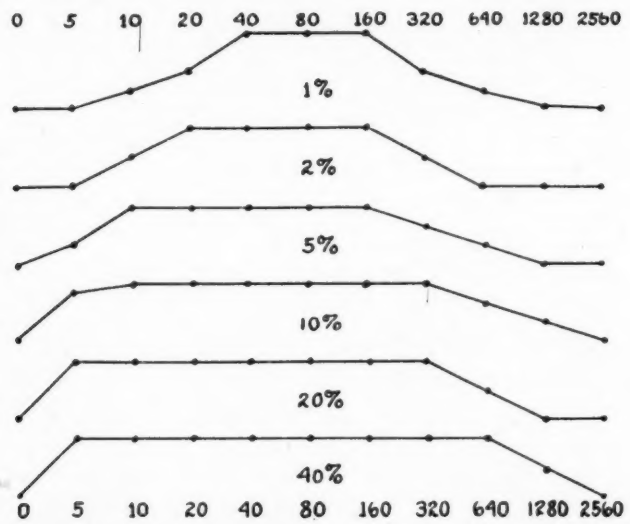
The most complete report is that of Hoffmann,[11] who compared *di*-allyloxyethyl-*di*-phenyl amidine hydrochloride (diocaine), 0.2 per cent; cocaine, 4 per cent; alypin, 2 per cent; phenocaine, 2 per cent; pseudococaine, 4 per cent; pantocaine, 1 and 2 per cent, and larocaine,

4. Koch, J.: Deutsche med. Wchnschr. **57**:678, 1931.
5. Schildbach, O.: Zentralbl. f. Chir. **58**:3026, 1931.
6. Volkmann, J.: Zentralbl. f. Chir. **58**:3076, 1931.
7. Kemkes, H.: Zentralbl. f. Chir. **58**:591, 1931.
8. Braun, Heinrich: Local Anesthesia, translated by Percy Shields, Philadelphia, Lea & Febiger, 1914, p. 40.
9. Weiss, K. E.: München. med. Wchnschr. **78**:2198, 1931.
10. Balcarek, A.: Klin. Monatsbl. f. Augenh. **88**:527 (April) 1932.
11. Hoffmann, W.: Ztschr. f. Augenh. **78**:20 (July) 1932.

2 and 5 per cent. Frey's hairs [12] were used to measure the degree of anesthesia quantitatively. Hoffmann was able to correlate sensitivity of the cornea quantitatively with the hairs and certain practical usages. Thus a certain definite degree of anesthesia was needed for tonometry, for removal of embedded foreign bodies and for merely wiping a foreign body off the surface. His conclusions are complicated because of the numerous drugs used; however, important essentials may be stated as follows:

1. Phenocaine caused the most pain and cramping of the lid.

2. Pseudococaine and pantocaine gave the highest degree of anesthesia, lasting from two to seven minutes.



The peak of the curves indicates anesthesia for the 2 Gm. hair. The duration of the anesthesia is indicated in seconds.

3. Anesthesia produced by diocaine lasted only one minute.

4. Anesthesia produced by a 2 per cent solution of larocaine lasted four minutes, while that produced by a 5 per cent solution lasted from six to seven minutes, but to a less degree than that produced by pseudococaine and pantocaine.

5. Larocaine, 5 per cent, pseudococaine 4 per cent, and diocaine, 0.2 per cent, caused dilatation of the pupils after from one to one and one-half hours, the diameter reaching 5.5 mm.

6. None of the drugs influenced accommodation.

7. None of the drugs caused corneal clouding.

8. Larocaine had the least influence on the vascular system.

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12. Cited by Basler, A., in Abderhalden, Emil: Handbuch der biologischen Arbeitsmethoden, Berlin, Urban & Schwarzenberg, 1930, pt. 7, p. 526.

In a recent report on pantocaine Marx [13] characterized the properties of the drug under eight headings. It seems proper to repeat these for my study of larocaine:

1. Burning.
   The length of time burning continues after the deposit of the drops is in direct proportion to the percentage of the solution. The 1 per cent solution causes a burning sensation for fifteen seconds; the 5 per cent solution, for one-half minute, and the 20 per cent solution for two minutes.
2. Change in the conjunctival vessels.
   None even with the 20 per cent solution.
3. Change in:
   (*a*) Pupil. None after two hours with even the 20 per cent solution.
   (*b*) Accommodation. No change.
   (*c*) Width of palpebral fissure. No change.
4. Tension. No change.
5. Clouding of cornea. None.
6. Toxicity. None was found even with a solid substance in any of the persons tested.
7. Sterilizability. In any dilution no positive cultures were obtained after boiling for twenty minutes, and the efficacy of the solution was undiminished.
8. Duration and depth of anesthesia (chart).

The accompanying chart shows the results obtained on the normal human cornea with larocaine in the percentages of 1, 2, 5, 10, 20 and 40 as tested with Frey's hairs. The hairs were furnished by Prof. A. C. Ivy of the department of physiology of the Northwestern University Medical School. Only those curves obtained when the 2 Gm. hair was tested are shown; Hoffmann used this hair for excessively painful and embedded foreign bodies. It may also be stated that the powdered larocaine as supplied by the manufacturer was used for local application for conditions of the tear ducts without untoward results. The subcutaneous injection of the 5 per cent solution, in quantities up to 2 cc., caused no undesirable general or local reaction. Practical clinical use of the drug in many ocular procedures has been made at the Michael Reese Hospital with excellent results.

13. Marx, E.: Klin. Monatsbl. f. Augenh. **89**:209 (Aug.) 1932.

GENESIS OF THE REFRACTION OF THE HUMAN EYE

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The approach to the understanding of the genesis of the refraction of the human eye lies in a consideration of the phylogenetic, ontogenetic and finally the sociogenetic factors involved. It seems scarcely necessary to call attention to the fact that in the adaptive reactions of animals to their physical and biologic surroundings the eye, one of the principal organs connecting the living being with its environment, plays an important part. Assuming that the normal anatomic and refraction forms for a given species are those which the organism has acquired during its struggle for life in a given locality, one might expect uniformity in the refraction and appearance of the eyes of the group; for individuals with unadapted refraction must have been eliminated from life or from the locality in question. Actually, however, this uniformity is not quite rigid, because there are variable factors in a locality which leave their influence on the animal form—climatic, edaphic and biotic. Animal forms may be observed to change, to renew themselves and to replace one another, a phenomenon known to biologists under the term "succession." It is natural that ocular refraction should be subject to such changes. Thus, variable and ever changing surroundings produce variable refractions in animals.

During the growth of a human being changes in refraction occur which are based on several factors. The first of these is accommodation. This faculty is most powerful in early childhood and gradually wanes toward annihilation in senility. The cornea shows only a relatively small growth from the infantile stage to that of the adult, having to increase in size only by one fifth or one fourth to obtain the final value, and this is reached as early as the third year of life. The subsequent increase in the radius of curvature is practically negligible. However, individual variations in corneal curvature are large, and may amount to 10 diopters and more. The lens undergoes great variations during its developmental cycle. Paralleling the growth of the entire eyeball, the growth of the lens proceeds most rapidly during the first year, but in contrast to that of the cornea, it never ceases. The lens gradually increases in the equatorial direction and diminishes in its thickness and curvature, becoming less powerful as a refractor. The sagittal axis of the eye is subject to even greater changes, and these

vitally affect the refraction. The average sagittal diameter of the infant's eye is 17.5 mm.; that of the developed adult eye is from 23.8 to 24.3 mm., an increase in length of from 6.3 to 6.8 mm. This growth, unlike that of the cornea, proceeds until the end of the body's growth, but unlike that of the lens, it does not continue through life. The elongation of the anteroposterior axis proceeds irregularly, the increase being most intensive during the period of strongest growth of the whole organism. The growth of the cornea and of the lens tends to weaken the dioptric power, but the elongation of the eyeball compensates for this by receding the retina, bringing on a relative or an actual myopia.

Thus, the newly born infant is hyperopic; emmetropia and myopia are very rare exceptions. As the eyes grow, some, with the gradual weakening of hyperopia, attain emmetropia; while others pass over this boundary line and become myopic. When the growth of the body is completed and the eye acquires a more stable form (about the twentieth year of life) this early and often rapid alteration of refraction ceases. The majority of mankind remain within the range of hyperopia; the larger part of the remainder proves to be emmetropic, and the smaller part myopic.

Excessive elongation of the eyeball produces an axial myopia of a high degree. This, I believe, has for the most part nothing to do with the ontogenetic development of the eye but is pathologic.

The eye is largely predetemined by heredity, preserving in its different elements, and often in its whole, the features inherited from ancestors according to the rules of hereditary transmission. It is thus easy to understand the diversity of refractions among members of the same family as well as among mankind generally.

The question of "normal" refraction is not easily decided. If one were to consider the opticogeometric definition, anything to either side of emmetropia would be anomalous. But one must remember that refraction is a dynamic state, constantly restless. One "emmetropizes" the eyes, or tries to approximate emmetropia, when one looks far ahead, whereas in order to examine near objects one "myopizes" the eyes. Hence the optimum or "normal" refraction at a given moment depends on the size and distance of the object sighted. Biologically, the optico-physical classifications are significant in their relation to the functional capacity of the eye under working conditions in which the capacity is affected by changes connected with sharp deviations from emmetropia (Steiger). In other words, a refraction may be "normal" for one occupation and "abnormal" for another, placing normalcy on a functional or biologic basis.

The problem of the origin of myopia has occupied the major part of all controversies over the genesis of refraction. One school teaches that myopia develops as a result of the excessive growth of the eye

in its anteroposterior diameter under the influence of intensive near work. Thus, myopia is an acquired property; it is effected by external agents and is not connected with the ontogenesis of the eye. According to this view, myopia may be controlled and prevented by adequate hygienic measures, by elimination of the predisposing exogenous conditions. The other school holds that myopia is subject to the laws of ontogenesis, just as hyperopia and emmetropia are. The values of the basic refractive components are determined by heredity. The occurrence of extreme refractive deviations is due to the survival of nonadaptable mutations and their failure to be segregated. Thus, myopia is not an acquired property, but like other types of refraction (emmetropia and hyperopia) is inborn and cannot be prevented. Its control might be accomplished, not by hygienic, but by eugenic, measures.

The error of the partisans of the theory of acquired myopia consists in the absence of perspective, in a narrow observational field and a cramped biologic horizon. On the basis of a purely physical index, these authors have isolated forcibly out of all the types of refraction (which is a biologic process) just one sector—myopia. But man attains myopia, starting with hyperopia and passing through emmetropia. Under identical conditions of life and work some people become myopic, others emmetropic, and still others remain hyperopic. The same narrowing of the biologic horizon caused these authors to isolate artificially the period of time required for refractive changes to develop. Fascinated by the school period of human existence, they argued illogically "post hoc propter hoc." The particularly noticeable increase of refraction during the intensive growth of the organism, which corresponds in time with the period of school studies, was explained by the pernicious influence of near work. A broader historic horizon would have found expression in a less phylogenetically narrow exposition of the myopia problem. Better use of historical facts would have pointed the way to the proper understanding of the so-called "professional myopia" and prevented the theory that there is a "working myopia."

Variations in the refractive states during the different periods of life of a human being are ontogenetic expressions of the regularity of individual development. Variations in the refractive states of different persons are phylogenetic expressions of the development of the genus. All are different aspects of the same biogenetic law.

The dissemination of mankind over the whole globe, with adaptations to the various ecologic conditions, has had no influence on the refraction. This is in contrast to the case of animals, in which the refraction and the anatomy of the eye are so clearly dependent on the surrounding conditions. Man creates conditions of existence suitable to his organs and liberates his organs, the eye included, from the formative influences of his medium. This accounts for the uniformity in design in the optical

apparatus of men of different races and different localities, as well as the identical (still judged by scanty evidence) ontogenetic process of the development of refraction.

Therefore the wide range of refraction variants for man which may be observed within the limits of a single locality cannot be due to previously nonadaptable mutations becoming adaptable in certain localities. The explanation will be given in the succeeding paragraphs which deal with the sociogenesis of refraction.

Myopia has been considered a social disease. Its development has been connected by some authors with the weakening of the visual organ on the basis of poor social and hygienic conditions. Nearsightedness has been believed to be an evil, even a calamity, threatening the well-being of society. An extensive general inspection of students in Germany which revealed a great number of myopic persons among them, as well as the erroneous conception of myopia as a school disease produced by intensive near work, brought the wide circles not only of medical men but also of teachers and public men to the belief that this form of refraction is a dangerous disease which threatens the sight, hence, the working capacity of certain large groups.

Along with the organization of human societies on the economic basis, with the development of industrial relations among men, with the appearance of class divisions, new forces came into action. When considering the eye as an optical device, one finds in it phenomena that develop according to the laws of physical optics. When studying the eye as an organ of sense, as a part of the living organism, one sees the general biologic laws being revealed in the development and functioning of its refraction. Now while treating the eye as the most important working organ of society I shall try to reveal the results of the action of social forces.

This is a very difficult and quite new problem. The ontogenesis of refraction proceeds, as it were, before one's eyes. All are witnesses to this process; one may easily observe it and study it, collecting extensive material in this way. Observers disagree only about the causes of this process, about its being determined by endogenous or exogenous factors. Ontogenesis of refraction is the present tense.

Phylogenesis of refraction is the past perfect tense: It pertains to the far off prehistoric times. However, copying a historian, who makes his conclusions with regard to antiquity on the basis of archeologic findings and observations of wild tribes existing in the present period, one can reconstruct the stages of the phylogenetic development by studying among the still living zoologic species the representatives of the forms which have disappeared long ago.

The biogenetic law, according to which these stages proceed in ontogenesis, is of great assistance in this respect.

Sociogenesis of refraction is a young process, awaiting its results in the future. The period of the socially organized human life is still so short that the consequences of the action effected by social forces could not be very clearly revealed in such a conservative human organ as the organ of sight and its refraction. But some influence must be felt; one has only to look for its traces. The fact is that refraction as an index and a factor of the working capacity plays an outstanding part in the working activity of man.

In a primitive society with the simplest organization of labor, in hunting tribes, for instance, a natural selection prevails the same as in the animal world, which tends to preserve variations of refraction within adaptable limits. Persons with strong deviations toward a high degree of myopia or hyperopia stand a poor chance to survive and to bring up progeny. As far as it is possible to judge by a few statistical investigations of refraction among wild tribes, as well as in the communities with the simplest forms of agriculture and cattle-breeding, the refraction curves for such peoples are of a binomial character with a steep drop at both bends, the majority of cases (up to 90 per cent) being concentrated between hyperopia of 2 diopters and myopia of 1 diopter, with a rare occurrence of high degrees of ametropia.

In the highest orders of cultural development, with the division of labor that exists inside the firmly established social and economic orders, with the formation of classes (leading class, artisans, etc.) the work of the eye has to be changed, and more varying demands are made on the refraction. As was stated in a foregoing paragraph, nonadaptable mutations may become adaptable in the animal world provided they find a suitable place in nature, for "the world has a countless number of localities." Similarly, the developed cultural society has many spheres in which to apply various forms of labor. While in the primitive community consisting of huntsmen and fishermen there is no place for myopic persons or for those with sharp astigmatism, in a highly developed society with widely spread education, fine crafts, refined handiwork, etc., myopic eyes which prove to be adaptable to these forms of labor have found and find their use.

Such eyes not only have found their use but have proved to be even advantageous, thanks to their myopia, especially in those times when the use of glasses was unknown and only myopes could continue and develop their professional activity till extreme old age without handicap from presbyopia. In the feudal society and state, with the formation of stable classes, this process of segregation by refraction went on, and it promoted the formation of working groups with frequent myopia. City states of the antique world, urban centers of medieval Western Europe, with their animated commercial and industrial activities, were populated by myopic elements who were unfit for the military work

and like enterprises, but found work in these centers. Guild organizations not only provided them with a living but also, owing to the hereditary transmission of forms of labor from generation to generation and from father to son, fixed their myopia as a hereditary property.

This process of differentiation in society by refraction could be observed also in the upper leading classes of the feudal society, whose members were engaged in continuous wars, and who, during the periods of relative peace, busied themselves with military exercises, hunting and similar pleasures and sports. A myopic boy born in such a family revealed at an early age his inability to join in all these pursuits; therefore he was predestined to another career, which required schooling, the knowledge of reading and writing. Such people entered the clergy, or diplomacy, or became representatives of scholastic science and the like professions. Here myopia became an advantage. But its danger for military men is illustrated by the case of the Swedish king Gustavus Adolphus, a highly educated man and a remarkable general, who was killed in the battle of Lützen as a result of his myopia.

Such a differentiation occurred in various epochs of the world's history and in different countries of the world where state organizations with a strict class structure existed and prospered. Such was the situation in China, Japan, India, the Pharaohs' Egypt, in city states of ancient Greece, in the Roman republic and Roman empire and in Western Europe. The remarkable preciseness of the minute artistic and technical labor producing the countless articles of art and crafts, an astonishing number of which were discovered on excavating ancient ruins, might be explained only by a considerable number of myopic people having worked in the corresponding professions and spheres of activity.

However, the remains of dead cultures enable one only to make suppositions about the sociogenesis of refraction. Let one try to find more direct evidence to prove that forces were existing in previous times which contributed to a division of society according to a refraction index. Being saturated with heredity the eye, as one of the most important organs of an adaptable value, is rather conservative. There will be nothing audacious in an attempt to carry out a kind of an archeologic excavation with regard to the eye when one is seeking such evidence. In this case the ophthalmologist, like an archeologist, should go to the places where once on a time the ancient cultural states prospered. Here he must study the eyes of those population groups which are the descendants of the ancient society which created this culture. Such are the interesting results, puzzling for the ophthalmologist, which were obtained by investigating the refraction among some of the people living along the coasts of the Mediterranean Sea—South Italians, Arabs, Copts in Egypt, Jews. The enormous number of myopes among Copts, according to Meyerhoff, is likely to be due to the racial influence, since

they are almost entirely an illiterate people. But Copts are the direct descendants of the ancient Egyptians. Steiger's point of view is more plausible, that the high percentage of myopia among the ignorant, poorly educated native population of South Italy is due to the inheritance from the high cultures which were there replacing one another before and during the Middle Ages. It is enough to remember the epoch of the Great Greece of the antique world and the epoch of Frederick II Hohenstauffen in the thirteenth century. These cultures, these populous flourishing cities animated by industrious tradesmen, craftsmen, painters, clerks and the like, disappeared long ago. But the eyes of their descendants, one's contemporaries, which are reflecting the modern world, bear the impression of the light of the civilizations faded away.

Along with the destruction of the natural economy and the transition to the capitalistic order, the same process consisting in the augmentation of the urban population, in the formation of the industrial proletariat, was going on. In this process factors of segregation and professional distribution according to the property of refraction continued to act and they have not ceased to act up to the present time.

Such is the most natural sociogenetic explanation for the considerable percentage of myopes among the students of the high and middle class schools in Germany, among every profession.

But life does not stand still; it goes on and on. Economic forms, industrial relations are changing; the old orders of society become unstable. Superimposed social strata and streaks, petrified like geologic stratifications, are being blown off and broken under the pressure of volcanic masses coming from below. Upheavals are observed, layers come upside down—and as a result Oskar Heinonen reports from Sweden a considerable decrease in the number of myopes among students of high schools owing to the change in the social structure of the student body, to the inflow of young people from the large popular masses. Moutinho and Mattos of Lisbon point out the same remarkable fact in Portugal, explaining it quite erroneously by the improved hygienic conditions. But in the course of discussion it was shown by their opponents that the true cause of this lies in the Portuguese revolution after which the lower classes of the population with a rare occurrence of myopia among them obtained access to university education.

There is hardly any need for more examples. Every ophthalmologist in the U. S. S. R. who has investigated the refraction among the pupils of the former colleges and among those of the modern schools is well acquainted with the enormous difference existing between the old and the new schools with regard to the numbers of myopes. The ocular complaints made by the majority of the students indicate the prevalence of hypermetropia among them. And the reason for this fact, still waiting to be investigated, is to be found in the social revolution.

When dealing with the sociogenesis of refraction one enters into a sphere of most complicated phenomena. First of all, the changes of refraction are not brought about directly by the social factors: The latter do not create new variants, but the formerly nonadaptable mutations (for instance, myopia of high and average degrees) may be assisted by these factors in spreading and taking root among certain groups of the population. The same social factors may enable the forms of refraction which are scarcely suitable for fine demands on the organ of sight (for example, astigmatism and hyperopia of high degrees) to exist and to survive. This process, in my country, in the socialistic society, is taken under strict control by means of professional segregation on the basis of the ocular refractions, for the sake of increased efficiency of labor and improved sanitation of the population.

Both in literature and in social intercourse one often meets with the question whether myopia is not the product of the highest culture, whether it is not the adaptation of the eye to the new working conditions which require intensive near work. According to my belief this question should be answered in the negative if it means that the assiduous work accompanied by straining of the organ of sight brought forth a special type of refraction adapted to such work.

Man as a member of a society in which the biologic law of the struggle for existence and of the survival of the fittest is not annihilated but only absorbed by the regular rules of social development takes possession of the natural forces by studying them and using them with the purpose of increasing the efficiency of labor. For this purpose man produces tools, beginning with the simplest instruments and ending with the most complicated machines. But first of all he tries to utilize and, if possible, to improve technically the working organs of his own body. Among these the organ of sight plays an outstanding part. Man has no control over the formation and development of the eye and its refraction. But he may exercise control over refractions from the economic point of view by correctly distributing types of refraction in the corresponding production processes, as well as by correcting the refraction variants by optical glasses.

Thus, in the modern cultural society, with great variations existing with regard to the division of labor, different types and degrees of refraction, predetermined ontogenetically, find their use. I do not refer here to the pathologic forms—these are the fields for the activity of medical science with its prophylaxis and therapy.

One more question among many problems untouched on—what is the future of refraction from the sociogenetic point of view? Phylogenesis resulted in the formation of this splendid organ of sight with its refraction. Ontogenesis shows the birth of the eye in the individual body, the process of its development and its wasting away with the

end of human existence: it indicates the course taken by refraction in this flow of the individual life.

In sociogenesis one observes the action of this organ, its refractions in the various processes of production and the distribution which that involves of the refraction variants among the different classes and working groups of the population. What will be the further direction taken by this distribution? Will new variants appear? Will nonadaptable mutations become adaptable in the future society? One cannot answer these questions. But one point remains clear: The unparalleled technical development armed all the working organs of the body with such a quantity of finest devices, refined the activity of the extrinsic sense organs, particularly those of sight and hearing, to such great extent, that all these organs became considerably free from the adapting effect of outward factors. Now a footless cripple can tear along in a car, while an armless cripple can control a complicated engine. Without straining one's sight one can see the minutest things, a thousand times less than a millimeter, as well as things situated a thousand kilometers from one.

Thus, liberated from the restraining influence of the ecologic factors, which are eliminated to a considerable extent by the culture of the modern society creating its own artificial medium, the biologically predetermined variability might become strongly revealed in refraction, giving rise to ever new variants of the latter.

It is almost impossible to describe anything new and original in this domain, which is already pierced in all directions by a series of investigators, and where manifold more or less profound and ingenious ideas have already been announced, and a countless number of hypotheses and theories built.

In 1922 after the war and revolution had cut off literary communication with Western Europe, I came across the book by Steiger, "Die Entstehung der spherischen Refractionen des menschlichen Auges," and I was astonished to see how his views on the genesis of refraction agreed with mine. In 1924 my point of view was stated in my article "The Problem of Myopia" in the *Russian Ophthalmologic Journal.* Now I am endeavoring to develop it.

This point of view, the result of thirty-three years of practice as an oculist, consists in the deep conviction that refraction, which seems to be the most immovable, stable and strictly physical part of the visual apparatus, is life itself and may be understood from the heights of general biologic contemplation.

Being one of the most important human working organs which help man to conquer nature by means of science and technic, the refractive apparatus determines by its types the fitness of man for any field of labor. Hence the action of social forces may be interpreted in the refractions of man as a member of an organized society.

ANALYSIS OF FIFTY-FIVE CASES OF TOBACCO-ALCOHOL AMBLYOPIA

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During the past eighteen months I have examined approximately 70 patients with tobacco-alcohol amblyopia, and from these I have chosen 55 for a more careful study. The latter were clinical patients at the Massachusetts Eye and Ear Infirmary and most of them I have examined frequently over a period of many months since they were first admitted to the clinic. This type of amblyopia continues to present many puzzling features, and it is almost as important for one to realize its unsolved problems as it is to know the clinical facts on which diagnosis, prognosis and treatment are based. It is especially important, when trying to evaluate the effect of some newly suggested treatment, to know what the normal course of the disease is and what may be expected without any therapy.

INCIDENCE

This type of amblyopia is seen in from about 0.3 to 0.5 per cent of all patients newly admitted to the eye clinic of the Massachusetts Eye and Ear Infirmary. Many older ophthalmologists feel that this condition is seen less frequently in this country now than formerly. Granting that this is perhaps true, there are, however, a large number of patients with the condition in whom it remains undiagnosed and who remain untreated for many months after the diagnosis should have been made if the oculist had used a tangent screen. During this period the vision, which might have been saved by abstinence, sometimes becomes irreparably impaired.

Males are much more frequently affected than females; only 2 of the 55 patients were females. Usher[1] found 27 females in a group of 1,100 patients with this disease, or 2.5 per cent. The average age of the present group was 55.6 years; the youngest patient was 25 and the oldest 77 years of age. Lautenbach,[2] in 1898, reported a case in a boy aged 13. The only description was this: "One of his cases was a boy aged 13 years who had all the typical symptoms of tobacco

This study was carried out at the Massachusetts Eye and Ear Infirmary, Boston, Mass.

1. Usher, C. H.: An Analysis of the Consumption of Tobacco and Alcohol in Cases of Tobacco Amblyopia, Ann. Eugenics **2**:245 (Oct.) 1927.

2. Lautenbach, L. J.: Ophth. Rec. **7**:419, 1898.

amblyopia and presented, as well, a tobacco heart and tobacco cachexia. After appropriate treatment and the removal of tobacco, the case improved." Such a description would not now be considered sufficient to classify this case as one of toxic amblyopia. Seventy-six per cent of the present series of patients were over 50 years of age.

In this series Italians were more often affected than persons of any other nationality. That this is due to an increased susceptibility in Italians seems unlikely, although there are references [3] to the comparative immunity from this disease enjoyed by the Turks and Spaniards; and, so far as I am aware, there is no evidence which disproves the possibility of a relative national immunity.

Over 20 per cent of these patients came more than 25 miles to the clinic, suggesting again that the diagnosis of the condition is being overlooked by certain practitioners, especially in the smaller communities.

A careful history concerning occupational hazards is, of course, essential in every case of amblyopia of undetermined origin. There was a great diversity of occupations among the patients who were employed, and in no instance did the amblyopia appear to be related to the patient's work. It was interesting to note not only that a large percentage were unemployed (47 per cent), but also that many of these stated that they had increased their use of tobacco and alcohol, or both, since they had stopped working. Traquair [4] stated that in England "the patients suffering from amblyopia (tobacco) belong almost entirely to the respectable working class, and the kind of individual who may be regarded as a 'soaker' or 'waster' is seldom found amongst them." In this country one sees many who are both "wasters" and "soakers," and not a few in the present series might be so classified. However, the disease occurs in all social classes.

Whether or not there is a seasonal incidence of the onset of this condition is not definitely known. Usually more of the patients are admitted to the clinics in the spring and summer months than during the winter,[5] but an accurate history as to the time of onset of the symptoms is not frequently obtained from clinic patients. The duration of symptoms before the patient comes to the oculist varies greatly (table 1). The evidence is inconclusive as to whether or not more cases develop from January to May, as table 1 suggests; one reason, at least, why these patients wait until spring and summer to come to the

3. de Schweinitz, G. E.: The Toxic Amblyopias, Philadelphia, Lea Brothers & Co., 1896.

4. Traquair, H. M.: Lancet **2**:1173, 1928.

5. Cossu, A.: Amblyopia alcoholico-nicotinica, Boll. d'ocul. **2**:297, 1923; quoted by Ascher in Zentralbl. f. d. ges. Ophth. **11**:294, 1924. Usher.[1]

clinic is, as Usher mentioned, that a centrocecal scotoma is much more annoying on sunny days when the pupils are contracted than when the illumination is less bright.

SYMPTOMATOLOGY

Usually this condition comes on very gradually, but infrequently the patients give the history of a rather sudden onset. How long it actually takes for the condition to develop is unknown, but 2 cases from this series are somewhat instructive in that respect.

An Italian laborer, aged 38, was examined as to refraction, and vision of 20/20 was obtained in each eye without a glass; forty-nine days later he returned, and vision was 10/200 in the right eye and 20/100 in the left; the visual fields were typical. The patient stopped smoking, and in two weeks vision was 20/50 and 20/40; in five weeks it was 20/30 in each eye, with characteristic visual fields. I saw the patient again eight months after the amblyopia was first detected, and vision was 20/20 in each eye.

TABLE 1.—*Duration of Disease Before Admission to the Clinic in Thirty-Eight Cases (from History)*

Duration, Mos.:	Cases
1 to 3	11
4 to 6	13
7 to 12	10
Over 12	4
Longest, 8 yrs.	
Shortest, 3 wks.	
Month of onset (from History):	
Jan. to May (5 mos.)	20
June to Dec. (7 mos.)	15

Another patient was followed twenty-six months. He was an unemployed man, aged 67, who when first seen had vision of 20/20 in each eye; in eight months vision had become 20/70 and 20/30; in thirteen months, 20/100 and 20/70; in fourteen months, 20/100 and 20/100. He finally took the advice to discontinue the use of alcohol, but decreased only slightly his use of tobacco to 4 oz. (113 Gm.) of Edgewood tobacco and 2 cigars weekly. When seen twenty-two months after the first examination he had vision of 20/40 and 20/40, and four months later it was 20/30 and 20/40. He still smoked the same amount of tobacco.

The complaints of patients with this condition are such as would be expected because of the scotoma. They state that there is a "fog" or "mist" in front of everything; that they can see other people all right except for the "haze" before their faces; that they see best in the early evening or even in the early morning before there is bright sunshine. Red is especially difficult for them to see, and they have trouble distinguishing pennies from dimes. Often their friends appear to them paler than usual, and they sometimes complain that their wives have lost their pink cheeks. Usually they have trouble with reading, but are able to perform household duties or manual labor without diffi-

culty. Illiterate patients and those who seldom read complain surprisingly little about their symptoms. Patients who, despite cecotemporal scotomas, have sufficient visual acuity to read books usually can read better with the left eye than with the right, because in a person reading from left to right this type of scotoma is less troublesome for the left eye.

Most of the patients in this series when they were first seen in the clinic had vision (table 2) of 20/200 or worse, although some had vision of 20/20 in each eye. This is important to remember, because the patients with good visual acuity do sometimes complain of marked blurring of vision due to the scotomas, and if the oculist does not carry out some sort of studies with the tangent screen he will probably fail to appreciate the nature of the case. Usually vision in the two eyes is about the same, but there may be a considerable difference, and vision may improve much more quickly in one eye than in the other.

TABLE 2.—*Vision at Time of Admission to the Clinic*

	Cases
1/200 to 10/200	16
11/200 to 20/200	21
20/100	2
20/70	6
20/50	4
20/40	2
20/30	4
Lowest, 1/200, 2/200	
Best, 20/20, 20/20	

There is some dispute as to whether the changes in the visual fields produced by this disease are pathognomonic. Hirshberg [5] attempted to distinguish alcohol amblyopia from tobacco amblyopia on the basis of the changes in the visual fields, but other observers have been unable to substantiate his work. Traquair,[6] in describing the visual fields in tobacco amblyopia, wrote that the "characters described, regarded collectively, do not appear to occur in any other form of amblyopia," and "the demonstration of typical defects enables a diagnosis to be made with confidence." De Schweinitz expressed a similar opinion. Lillie,[7] on the other hand, maintained that he had seen numerous other conditions, for example, multiple sclerosis and lead poisoning, which had produced changes in the visual fields similar to those occurring in tobacco amblyopia.

In the present series centrocecal scotomas were noted, usually much larger for red than for blue, with sloping edges, and one or more dense areas (nuclei) between the blindspot and the fixation area. The

6. Traquair, H. M.: An Introduction to Clinical Perimetry, St. Louis, C. V. Mosby Company, 1933.

7. Lillie, W. I.: Am. J. Ophth. **17**:110, 1934.

centrocecal or cecotemporal characteristic of the scotoma was most striking. A nucleus in this area for an angle as large as 45/1000, when a white object was used, was found in few cases. One of these patients had a visual acuity of 20/20 in each eye with the Snellen test type despite this scotoma. Another patient with a similar large dense nucleus, who had been intensively studied by the neurological service before I saw him was followed many months and made an excellent recovery. With a 3/330 angle and a white test object, no peripheral contraction of the visual fields was noted in any patient, so that if there was any general depression of the whole field it must have been slight. In no case was there a scotoma in only one eye, although it was sometimes much more marked in one eye than in the other.

One patient was definitely known to have had red-green blindness sixteen years previous to application for treatment because of amblyopia. At the time of examination he showed red-green blindness, and

TABLE 3.—*Ocular Pathology*

Lesion	Cases
Lenticular opacities	7
Vitreous opacities	4
Temporal pallor of disk	19
Grade 4 temporal pallor	2
Vision = 20/20 20/30, 20/20 20/40	
Macular changes	16
Retinal hemorrhages	2
Sclerosis	8
Old exudates	2
Drusen	2

the color fields were of no value. Another patient, however, who had previously been a motorman on the subway cars and had passed the tests for color blindness without difficulty, lost all perception of red even in the nasal field. Groenouw [6] stated that such a finding was possible; however, it must be rare.

PATHOLOGY

The amount of pathologic change found in ocular structures anterior to the disk and retina is no more than may be expected from any equally large group of patients with the same age distribution. The same may be said about the number of these patients showing retinal angiosclerosis, exudates, hemorrhages or drusen. I know of no good evidence for attributing the angiosclerosis or the hemorrhages to the alcohol or tobacco.[8] General pathologists at the Boston City Hospital who for many years have been performing autopsies on patients with chronic alcoholism feel sure that patients of this type show less, rather

8. Zentmayer, William: Am. J. Ophth. **8**:365, 1925.

than more, arteriosclerosis than others of the same age. Henry A. Christian,[9] Physician-in-Chief, Peter Bent Brigham Hospital, Boston, writes: "There is no definite evidence that alcohol in any way is a cause of cardiovascular diseases or hypertension." Two patients from the present series showed retinal hemorrhages: One, aged 60, showed a deep retinal hemorrhage along the course of the inferior temporal vessels; the other, aged 68, had a small hemorrhage in the nerve fiber layer. Certainly, I saw no unusual amount of retinal angiosclerosis, and at the medical clinic at the Massachusetts General Hospital generalized arteriosclerosis and hypertension were reported in only 4 of 22 cases.

The optic disks may show no changes at any time during the course of the disease. A definite pallor of the temporal side of the disk in the area occupied by fibers of the papillomacular bundle was noted in 19 patients. Once this pallor becomes manifest it never disappears although the patient's vision and fields become normal. The degree of pallor does not necessarily bear any relation to the visual acuity or the visual fields, and of the 2 patients who had the most marked temporal pallor one after many months had vision of 20/20 in each eye and no field changes, and the other had vision of 20/30 in the right and 20/40 in the left eye, with a slight centrocecal scotoma for red.

Vogt[10] described ophthalmoscopic changes between the disk and the fovea. Using red-free light, he found that the appearance of the nerve fiber pattern between the papilla and the macula was altered. Many of the patients in this series failed to show a foveal reflex, and these are included among the 16 mentioned in table 3 as having macular changes. Of course, it is not infrequent to find an absence of the foveal reflex in patients of this age, so that too much emphasis should not be placed on that finding. However, some of the patients do show a faint, fine mottling in the macular area which may be of some significance, but it is most important to distinguish these findings from those of senile macular degeneration. Several of the present series had received this diagnosis when examined ophthalmoscopically elsewhere. All of these patients showed typical visual fields and improved after abstinence, which proved that they had toxic amblyopia. As is well known, elderly patients may have a marked stippling or a fine mottling in the macular area with no decrease in vision; and if in such cases amblyopia does result, studies with the tangent screen are most advisable before dismissing the cause of the poor vision as early senile degeneration.

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9. Christian, H. A.: Alcohol and Man, New York, The Macmillan Company, 1932.

10. Vogt, A.: Die Nervenfaserzeichnung der menschlichen Netzhaut im rotfreien Licht, Klin. Monatsbl. f. Augenh. **66**:78, 1921.

ETIOLOGY

The types of alcoholic beverage used by 37 patients are given in table 4. One may see that in this series the use of wine, usually of the home-made variety, was associated with this disease most frequently, although it is probable that any form of drink containing ethyl alcohol may produce it. There is no evidence that one is dealing with methyl alcohol. Many of the Italian patients had been drinking with their meals the same amount of the same kind of wine for many years; in no instance were there 2 cases in the same family although other members of the household, older as well as younger, often drank equally large amounts.

TABLE 4.—*Types of Alcoholic Beverage Used*

Beverage	Cases
Wine	12
Alcohol	7
Beer	5
Whisky	3
Gin	1
Beer and wine	3
Beer and whisky	3
Wine and alcohol	1
Whisky and wine	1
	37

TABLE 5.—*Forms of Tobacco Used*

Form	Cases	Oz. per Wk (Av.)
Pipe tobacco	21	10.7
Italian stogies (28 stogies)	4	8.4
Cigarets (7 packages)	5	4.2
Cigars (23 ordinary)	3	6.9
Cigarets and cigars	1	5.1
Pipe and chewing tobacco	1	12.0
Pipe and cigars	2	5.3
Pipe, cigars and chewing tobacco	1	15.0
	38	
Average amount smoked		8.9

Table 5 indicates that pipe-smokers get this disease most frequently. Usher [1] reported 4 cases in which cigarets only were smoked. Contrary to what may be thought, the percentage of nicotine in some cigarets is reported [11] as higher than that in some pipe tobacco; i. e., the nicotine content of Prince Albert Tobacco is 1.82 per cent; that of Chesterfield cigarets, 2.53 per cent. However, there are many other factors besides the nicotine content of tobacco; it need only be mentioned here that the form in which the tobacco is used is important. A cigaret will tend

11. Connecticut Agric. Exper. Stat. Bull., nos. 295 and 307, 1929; cited in Nicotine Content of Tobacco: A Re-Publication of Some Interesting Figures, Bureau of Investigation, J. A. M. A. **101**:385 (July 29) 1933.

to yield less nicotine and other volatile substances in the tobacco smoke than a thick cigar even though the latter may have a lower nicotine content in the dry state. This is partly because in the former there is more complete combustion and much of the volatile nicotine has an opportunity to condense before reaching the mouth of the smoker. Most of the nicotine is found in the cigaret stub.

Table 6 is interesting in several respects. It indicates that the heavy drinkers tend also to be heavy smokers, and that some of those who used the least alcohol also used comparatively small amounts of tobacco. In my experience it is often impossible to tell whether the amblyopia is due principally to the alcohol or to the tobacco. Either may produce it alone, but most frequently they are both used by the patient. Careful questioning at every visit revealed that almost every patient had used both alcohol and tobacco, although sometimes one was used much more than the other. A case probably illustrating the effect of tobacco was

TABLE 6.—*Amounts of Alcohol and Tobacco Consumed*

Alcohol Consumed, Oz. per Week (Av.)	Patients	Tobacco Consumed, Oz. per Week (Av.)
0–2 (0–59.1 cc.)	5	8.1 (252 Gm.)
2–4 (59.1–118.2 cc.)	4	4.5 (140 Gm.)
4–8 (118.2–236.6 cc.)	6	10.0 (311 Gm.)
8–16 (236.6–473.1 cc.)	1	6.3 (196 Gm.)
16–32 (473.1–946.3 cc.)	8	8.7 (270 Gm.)
32–64 (946.3–1892.6 cc.)	9	10.8 (335.59 Gm.)
Over 64 (1892.6 cc.)	4	12.8 (398 Gm.)
Patients with history thought reliable		37
Ounces of alcohol consumed (av.)		28.6 (890 Gm.)

that of a 77 year old Irishman who drank 3½ quarts (3.3 liters) of whisky and smoked 2 oz. (62 Gm.) of Sensible Tobacco weekly. Four months after his first visit his vision had improved from 5/200, 6/200 to 20/40, 20/50, and when heard from after eleven months he had no visual complaints. He insisted that he had not and did not intend to decrease his alcoholic intake, but that he had used no tobacco since abstinence was advised. On the other hand, consider a patient who continued to smoke but stopped the drinking of alcoholic liquors. He was a 44 year old Negro, a porter, who had been drinking 2½ quarts (2.3 liters) of alcoholic liquors and smoking 14 packages of cigarets weekly. When he was seen again in three months his vision had changed from 2/200 in each eye with a large typical scotoma to 20/20 in each eye and no scotoma; he had stopped imbibing all alcoholic beverages but continued to smoke exactly the same amount.

The idea is still prevalent that these patients have to smoke or drink excessively before getting the disease; this is not true, unless by "excessively" one means that the intake is too great for that particular patient. Some of these patients use only small amounts of alcohol and tobacco

and I think there can be no doubt that the increased susceptibility of the patient is a far more important factor than the amount consumed. Usher,[1] in an interesting analysis of 1,100 cases of tobacco amblyopia and 500 control cases, found that of the persons over 50 years of age the controls used more tobacco than the patients with amblyopia.

Previous studies [12] have indicated that the same agents which precipitate the toxic amblyopia also bring about changes elsewhere in the body, and this suggests the idea that the amblyopia is merely the localized manifestation of a mild generalized intoxication. It would be most desirable to discover what causes the decreased resistance in the patients. Extensive examinations were performed in the neurologic, medical, dental and otologic departments at the Massachusetts General Hospital. When indicated, examinations by consultants in other specialty clinics at that institution were made. Table 7 shows that the results of the neurologic examinations were usually essentially negative; peripheral

TABLE 7.—*Results of Neurologic, Dental and Medical Examinations*

Examination	Patients Examined	Patients Found Normal	Patients with Morbid Conditions
Neurologic	10	8	2 (1 patient, decrease of vibratory sense; 1, paralysis of arm)
Dental	28	5	23 (11 patients, apical abscesses; 10, diseased teeth, without roentgen examinations; 2, pus pockets)
Medical	22	11	11 (1 patient, pernicious anemia; 1, chronic nephritis; 1, pellagra; 2, faint trace of sugar; 4, arteriosclerosis and hypertension; 1, enlarged liver; 1, deficient in vitamin A)

neuritis was present in 1 case not included in this series. A great deal of dental trouble was revealed, but when one considers the great percentage of clinical patients who have diseased teeth the findings are perhaps of questionable significance. Some patients with marked dental infection showed a strikingly rapid return of normal vision after abstinence although they had refused dental treatment and the condition of the teeth was unchanged. However, many otologists feel that dental disease may cause toxic deafness, a condition sometimes associated with toxic amblyopia.[12a]

Half of the medical examinations gave negative results. Two patients had faint traces of sugar in the urine on one examination, and 1 patient had a decreased tolerance to sugar on test, but none had diabetes. However, Waite and Beetham [13] in a recent intensive study

12. (*a*) Carroll, F. D., and Ireland, P. E.: Association of Toxic Deafness with Toxic Amblyopia Due to Tobacco and Alcohol, Arch. Otolaryng. **21**:459 (April) 1935. (*b*) Carroll, F. D.: Cerebrospinal Fluid Studies in Ten Cases of Tobacco-Alcohol Amblyopia, Am. J. Ophth. **18**:720 (Aug.) 1935.

13. Waite, J. H., and Beetham, W. P.: New England J. Med. **212**:441, 1935.

of 2,000 diabetic patients found 7 who had tobacco-alcohol amblyopia and there seems to be general agreement that this condition occurs more frequently in diabetic than in nondiabetic persons. Two patients (table 8) had positive Wassermann reactions of the blood although syphilis had not been suspected.

Table 8 shows merely that patients may have syphilis as well as toxic amblyopia. Studies [12b] of the cerebrospinal fluid showed a slight but definite increase in the total protein content in some patients.

To rule out the possibility that the amblyopia was caused by any metal, i. e., lead, arsenic or copper, contaminating the alcohol or tobacco, Mr. Richardson, at the spectroscopy laboratory of the Massachusetts Institute of Technology, made spectrograms of the spinal fluid from 1 patient and of the blood from 6 patients. These spectrograms were normal. To determine whether the patients showed any cutaneous hypersensitivity to the tobacco which they were using, patch tests with the

TABLE 8.—*Data on Wassermann Tests*

Blood:	
Patients tested	33
Patients normal	31
Patients with positive reactions	2
Cerebrospinal Fluid:	
Patients studied	10
Patients with dynamics normal	10
Patients with less than 10 cells	9
Patients with total protein less than 40	4
Patients with total protein 40 or over	6
Patients with total protein 60 or over	2

patient's own tobacco and intradermal tests with tobacco extract were performed on a few of them. These tests were negative.

TREATMENT

The treatment recommended for all of the patients was abstinence, and the results (table 9) can be attributed very largely to this one factor. In these statistics a patient was ordinarily not considered to be improved unless he could read at least two more lines of letters on the Snellen test chart than at his first examination. A small number of patients who insisted on getting some medicine were given strychnine sulphate from 1/60 to 1/30 grain (0.001 to 0.002 Gm.) three times a day, but the course of the amblyopia in these patients seemed to be no different from that in patients who did not receive this drug. A few patients had possible foci of infection removed. Several patients were given a single inhalation of amyl nitrate, and 4 patients received a single injection of a solution containing sodium nitrite intravenously while they were seated in front of a test chart. One patient from the latter group seemed to have a slight temporary improvement in vision,

TABLE 9.—*Progress of Patients*

Period of Observation, Mos.	Patients Seen	Patients Improved				Patients Not Improved				Patients Worse		Summary
		Total	Abstained	Decreased Intake	Did Not Decrease Intake	Total	Abstained	Decreased Intake	Did Not Decrease Intake	Total	Did Not Abstain	
2	17	7*	6	..	..	10†	3	..	4	..	..	Of the 9 who abstained, 6 improved; of the 4 who did not decrease intake, none improved
4	16	6	2	4	..	10	2	2	6	..	..	Of the 4 who abstained, 2 improved; of the 6 who decreased intake, 4 improved; of the 6 who did not decrease intake, none improved
6	10	5*	3	..	1	3*	..	..	2	2	2	Of the 3 who abstained, 3 improved; of the 5 who did not decrease intake, 1 improved and 2 were worse
9	10	7	5	2	..	2	..	..	2	1	1	Of the 5 who abstained, 5 improved; of the 2 who decreased intake, 2 improved; of the 3 who did not decrease intake, none improved and 1 was worse
12	6	4	2	1	1	2	..	..	2	..	..	Of the 2 who abstained, 2 improved; the 1 who decreased intake improved; of the 3 who did not decrease intake, 2 did not improve
More than 12 (av. 20)	13	10	1	7	2	2	..	..	2	1	1	The 1 who abstained improved; of the 7 who decreased intake, 7 improved; of the 5 who did not decrease intake 2 improved

* In 1 case it is not known whether the patient decreased intake.
† In 3 cases it is not known whether the patient decreased intake.

but there was no apparent change in the others. A study is now being made of the effect of diet in the treatment of this condition. Some observers [14] have suggested that "alcoholic" polyneuritis is intimately associated with a vitamin deficiency (B) rather than due to direct action of alcohol on the nerves.

There can be little doubt about the importance of abstinence in the treatment of this condition. However, it cannot be denied that some of these patients improved who only slightly decreased their intake, and very infrequently a patient who continued to smoke and drink as much as before the development of the amblyopia experienced a return of normal vision.

CASE 1.—A 58 year old Irish freight handler was first seen when he had vision of 20/70, 20/40. It was impossible to examine the visual fields satisfactorily at that time, although a typical centrocecal defect was later obtained, but he did have a central scotoma for color. He had been drinking large amounts of alcoholic beverages and smoking 14 ounces (435.4 Gm.) of Sensible Tobacco weekly. At three months vision was 20/50, 20/40; at six months, 4/200, 5/200. The Wassermann reaction of the blood was negative. The patient continued to smoke and drink. At seven months vision was 4/200, 4/200. The cerebrospinal fluid was normal except for a protein content of 60 mg. per hundred cubic centimeters. At eight months vision was 6/200, 8/200; at nine months, the same; at ten months, 20/200, 8/200; at ten and a half months, 20/100 in each eye; at eighteen months, 20/30 in each eye. The man was then drinking 1 quart (500 cc.) of beer and "1 shot of hard stuff" daily, as well as smoking from 3 to 4 pipefuls of Sensible Tobacco. At twenty-five months vision was 20/40, 20/40. The patient was drinking from 7 to 10 quarts (6.5 to 9.5 liters) of beer and smoking 8 ounces (249 Gm.) of Sensible Tobacco weekly. At twenty-six months vision was 20/20, 20/20; he was using 6 quarts (5.5 liters) of beer and 2 ounces (62 Gm.) of tobacco weekly.

TABLE 10.—*Summary*

Abstainers	1. Improving	18
	2. Not improving	1
Nonabstainers	1. Improving	3
	2. Not improving	14

CASE 2.—A 68 year old unemployed Italian had vision of 5/200, 3/200, and typical visual fields when first seen. He was consuming 14 glasses of beer, 3½ quarts (3.3 liters) of wine and 6 ounces (187 Gm.) of Mayo Tobacco weekly. X-ray pictures of the teeth and sinuses showed nothing of importance except some thickening of the lining of the sinuses; the Wassermann reaction of the blood was negative. At three months vision was 20/50, 20/70; at four months, 20/30, 20/40; at twenty-four months 20/20, 20/20. The man was still drinking the same amount of beer and wine, but had decreased his use of tobacco to 3 ounces (93 Gm.) of Mayo Tobacco weekly.

14. Minot, G. R.; Strauss, M. B., and Cobb, S.: New England J. Med. **208**: 1244, 1933.

Table 10 indicates the value of abstinence. Included among the abstainers in this table are those who markedly decreased their use of tobacco and alcohol and among the nonabstainers some who slightly decreased their consumption of these agents, but the evidence is strikingly in favor of abstinence as the potent factor in therapy.

PROGNOSIS

The prognosis is a difficult subject. Old patients frequently get well as quickly as the young. Patients with marked pallor of the disks temporally and even a very shallow excavation in this area, signs ordinarily considered diagnostic of optic atrophy, may obtain vision of 20/20 for each eye, with no defects in the visual fields. Infrequently vision may get worse for a time even after stopping the toxic agents. Sometimes, despite abstinence, the patient shows no improvement for many months and then vision returns in a surprising manner. During this period the visual fields may or may not be improving, and often after the visual acuity becomes normal there is a residual scotoma between the blindspot and the fixation area.

In general, if the patient abstains, the prognosis is very favorable. However, it is impossible to foretell which patient will have a return of normal vision and which one will have only a partial improvement. One 40 year old woman who had perfectly normal appearing disks and vision of 20/200 on her first visit abstained, as testified to by several members of her family, from all alcohol and tobacco for thirteen months, but her vision improved to only 20/70, 20/50, and the temporal side of the optic disks became progressively paler. On the other hand, a 59 year old Italian was first examined when he had vision of 20/200 in each eye and typical scotomas. He abstained for four months, but his vision did not improve, so he started drinking and smoking again. When seen two years after the first visit his vision had improved to 20/50 in each eye; but he was still using 7 quarts (6.5 liters) of home-made wine, 28 glasses of beer, ½ pint (250 cc.) of whisky, 2 ounces (62 Gm.) of Mayo Tobacco and 14 Italian cigars weekly. Certainly there are important factors in the causation and progress of this disease besides tobacco and alcohol, but until more is known about them abstinence must be strongly advised.

SUMMARY

Fifty-five cases of toxic amblyopia associated with the use of tobacco and alcohol were studied. It is felt that the diagnosis could be made earlier and more often if studies with the tangent screen were performed more frequently. The condition should not be confused with early senile macular degeneration. The amblyopia may develop after the use of only moderate quantities of tobacco and alcohol, and these

substances should be considered as only two factors which, when combined with others, bring about the disease.

Extensive medical, neurologic, dental and otologic examinations were carried out on the patients, and the results are presented. The progress of the disease was followed by numerous ophthalmic reexaminations over a period of many months, and the relation of abstinence to improvement was especially noted. The value of abstinence is stressed, but cases are cited in which the patients improved without markedly decreasing their use of tobacco and alcohol. There is great variability in the normal course of the disease, and this should be considered in attempting to evaluate the effect of any treatment.

FILARIA SUBCONJUNCTIVALIS

REPORT OF A CASE

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Filaria loa, a nematode peculiar to Africa, is rarely found in this country. Because of this fact, and by virtue of the startling appearance of the parasite under the bulbar conjunctiva, the following case may deserve detailed commentation.

REPORT OF A CASE

History.—M. L., a white man, aged 48, was admitted to the clinic of Dr. Bernard Samuels at the New York Eye and Ear Infirmary with the sole complaint that he had "a worm in the right eye." The patient was of French extraction, an inhabitant of Africa, and a sailor by trade. He stated that he had suffered from malaria in 1914 and amebic dysentery in 1918. No other illnesses were recalled. He denied excessive use of alcohol, drugs or tobacco. During 1920 he was employed in the French Cameroons as a hunter of mahogany. On his trips into the interior he drank only filtered water and took a daily dose of 5 grains (0.32 Gm.) of quinine. He was bitten by the tsetse fly, which in that area is innocuous. From this period until 1928 he was not living in the area in which infestation with Filaria loa is endemic, nor was he aware of the presence of this parasite in his body.

In 1928 he was awakened at night by the sensation of a foreign body in his right eye. While trying to locate the irritant with the aid of a mirror, he saw the parasite lying under the bulbar conjunctiva. Coincident with this discovery, he noticed a lump several inches in diameter on the flexor surface of his right forearm. It was not discolored, but was warm to the touch and was accompanied by an itching sensation. When a physician was reached the next day, both the worm and the swelling had disappeared, not to be seen again until 1931.

During the interim, while he was in Italy, syphilis was diagnosed and a year's course of treatment was undergone. In 1931 the parasite again made its appearance in the right eye. The patient was seen at the New York Eye and Ear Infirmary, but the parasite had disappeared into the orbital tissue before it could be removed. No Calabar swellings were noticed. Examination of the blood by one of us (Mr. Burchell) revealed many microfilariae. The most recent appearance of the parasite was in December 1934, when it was removed from the bulbar conjunctiva of the right eye. Calabar swellings were again absent. Subjectively

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the patient complained only of a foreign body that seemed to shift its position in the eye.

Physical Examination.—The patient was 5 feet, 8 inches (172.7 cm.) in height, weighed 140 pounds (63.5 Kg.), and was of ruddy complexion and nervous temperament. General examination revealed no abnormalities. The blood pressure was 140 systolic and 90 diastolic, and the pulse rate was 74 per minute. Under the bulbar conjunctiva of the right eye was a wriggling worm. The overlying tissues were not congested, nor was there surrounding edema. The fellow eye was normal. Each eye showed the lens and media to be clear, and nothing unusual was seen in the fundus. Vision, without correction, was 20/30 with each eye.

Laboratory Examination.—The specific gravity of the urine was 1.010. The urine was light yellow. No albumin, cells or casts were found. There was no chyluria. The blood count showed 4,800,000 red cells per cubic millimeter, with

Fig. 1.—*A*, eye with Filaria loa under the bulbar conjunctiva. *B*, adult filaria removed from the conjunctiva.

a hemoglobin content of 92 per cent (Sahli); the percentage of the red cells was 96, and the color index was 0.9. The platelets were abundant. The total number of white cells was 16,750; the differential count showed the lymphocytes to be 34 per cent, monocytes 3 per cent, polymorphonuclear leukocytes 50 per cent, of which 47 per cent were segmented, and eosinophils 13 per cent. From 1 to 4 microfilariae were encountered in each drop of whole blood. The sedimentation time was slightly active with an index of 18 in sixty minutes. The Wassermann test was positive with both antigens, also the Kahn test. During the day, many microfilariae were found in every drop of blood; at night the blood showed none. Attempts to produce the disease experimentally in the guinea-pig and the rabbit were unsuccessful. The microfilariae in a blood smear, with the edges of the cover glass sealed with paraffin, lived from eight to fourteen days in an incubator maintained at body temperature.

Treatment.—The nematode was grasped through the bulbar conjunctiva with the Lester forceps, after which a 2 per cent solution of cocaine was instilled. When the anesthesia was complete, a small opening was made through the conjunctiva near one extremity of the worm with the Stevens scissors. The worm was then grasped with another pair of forceps and delivered from its bed by winding it on the latter instrument. The eye was left uncovered, and by the next day the wound had closed. There was no bleeding. The adult filaria removed was 3 inches (7.6 cm.) in length and 1/10 inch (0.16 cm.) in diameter. Systemically, a course of intramuscular injections of fuadin, a sodium antimony salt, was given.

HISTORICAL REVIEW

That a filaria occurs under the conjunctiva has been known since the end of the sixteenth century. A print made in 1597 seems to show an operation for its removal.[1] It has been known by various names, but the one, "Loa loa," given it by Guyot in 1778 is used today. This is a combination of the name of the genus, Loa, and the name of the species, loa, of the phylum to which it belongs. Some of the synonyms are "Filaria occuli" (Gervais and Van Beneden, 1859), "Dracunculus occuli" (Deising, 1860), "Dracunculus loa" (Cobbold, 1864) and "Filaria subconjunctivalis" (Guyon, 1864).[2] Microfilaria diurna and Loa loa are different stages in the life cycle of the same parasite.[1]

EPIDEMIOLOGY

Loa loa is endemic along the west coast of Africa from Benguela to Sierra Leone. It is most common in Old Calabar, The Cameroons and along the Ogomé River. Its presence has been recorded in the interior as far as 600 miles from the coast. The cases reported from India are of doubtful validity, while those found in the West Indies and South America appear to have been imported from West Africa.[2] Both Loa loa and Filaria perstans are fairly common in the western part of Gabon, West Africa. Microfilaria diurna was found in the blood of 10.38 per cent of 880 natives examined.[3] The zoological distribution of Loa loa is confined almost entirely to man, but, according to Plehor,[4] the natives of The Cameroons assert that it occurs in goats and sheep.

The parasite infects man through the bite of the mangrove fly, although Blanchard [1] stated that the worm is taken into the body through the drinking water. Much doubt has been raised as to the life cycle of

1. Reference Handbook of Medical Sciences, New York, William Wood & Company, 1903, vol. 6, pp. 201 and 212.

2. Castellani, A., and Chalmers, A. J.: Manual of Tropical Medicine, ed. 3, London, Baillière, Tindall & Co., 1919, pp. 645, 646 and 647.

3. Galliard, H.: L'éosinophilie sanguine au cours des filarioses chez les indigènes du Gabon, Sang **7**:711, 1933.

4. Plehor, cited in Manson, p. 761.

Loa loa and its intermediary host, but in 1929 Faust [5] gave the following review:

The life cycle of Loa loa involves certain species of mangrove flies (Chrysops dimidiata, Chrysops silacea and possibly other species of this genus) which are day feeders. As early as 1895 Manson suggested on epidemiological grounds that Chrysops Dimidiata was the intermediary host of the infection. The work of Leiper showed that other biting insects were unsuitable hosts. The microfilaria are taken into the stomach of Chrysops when the fly bites a patient harboring the microfilariae in his peripheral blood. The larvae develop and then migrate to the head where the mature ones may be found in the largest numbers on the tenth day. Their numbers in highly infective flies amount to several hundred. While most of the larvae leave their dipteran host in one migration, the fly may remain infective for a period of five days. Within sixty seconds after the worms have emerged from the fly, they have disappeared under the skin of the human host. Attempts to infect monkeys, rabbits, and guinea-pigs have been unsuccessful, although the larvae readily penetrate the skin of the guinea-pig. Nothing is known of the development of the worms, once they have reached the subcutaneous areas of the human host. The life of the adult worm in man may be as much as fifteen years or more.

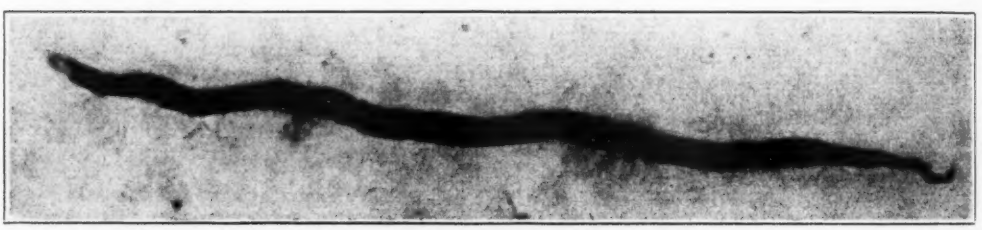

Fig. 2.—Photomicrograph of Microfilaria diurna.

It is believed that the embryos in the human host escape from the adult "mother" and pass via the lymph spaces into the blood stream where they are known as the microfilariae diurnae. They measure 298 by 7.5 microns. These are found in the circulating blood only in the daytime and are not affected by any alteration of the habits of the host.[2] The microfilariae are first seen in the peripheral circulation at 8 a. m. They reach their peaks in number at 1 and 5 p. m., and they are not to be found after 10 p. m.[6]

THE PARASITE

The adult worm measures from 3 to 15 cm. in length and has a maximum breadth of from 0.3 to 0.4 mm. The posterior part tapers gradually toward the tail, which is characterized by the presence of

5. Faust, E. C.: Human Helminthology, Philadelphia, Lea & Febiger, 1929, p. 464.

6. Fabre, J. A. J.: Essai sur la filaire de l'oeil humain (loa loa Guyot 1778), Paris, Amédée Legrand, 1924.

many small round protuberances of the cuticle that vary in number, size and arrangement. These are more numerous in the female. The body is filiform, cylindric, whitish and semitransparent. Microfilaria diurna is similar in size and structure to Microfilaria Bancrofti. It is enclosed in a sheath; its tail is pointed and has the same V-shaped spots. It is practically impossible to distinguish the two in living blood specimens. In dry smears Microfilaria diurna assumes a stiff angular attitude in contrast to the graceful curves of Microfilaria Bancrofti. The tail is usually coiled.[7]

PATHOGENICITY

The adult worm may be noted under the skin of the finger, back, breast, scrotum, penis, eyelid, conjunctiva and mucosa of the tongue. It moves quickly. It may cause an itching, creeping sensation, an irritation of the parts involved, an occasional lancinating pain and, elsewhere on the body, Calabar swellings. These are smooth, slightly raised tumors, temporary in character. They are seen on the head, arms and legs. They are caused by the presence of the parasite (Guyot, 1778) [2] or as the result of a reaction to toxins produced by it (Low [8]). The symptoms are in general benign, but Neumann [9] reported a case in which abscesses developed at the site of the Calabar swellings.

DIAGNOSIS

The diagnosis is dependent on the recognition of the microfilaria in the blood and on the determination of its cycle of appearance. An adult worm should be found. It may be brought to the surface by the application of heat. Calabar swellings are present, or a history of them may be elicited. The fact that the patient has lived or has visited in the endemic area of Africa must be determined. The edema of the extremities that so often characterizes other types of filariasis is absent. The laboratory tests show an eosinophilia that may rise to 50 per cent. There is no chyluria.

TREATMENT

The natives drive the nematode from the eye with a grain of salt placed in the conjunctival sac, or they extract the worm with a thorn.[1] Leger and Queinnec [10] stated that many drugs have been used without

7. Manson, Patrick: Tropical Diseases, ed. 5, New York, Cassell & Co., 1914, p. 763.

8. Low, G. C.: Some Points in the Pathology of Filariasis, J. State Med. **39**: 594 (Oct.) 1931.

9. Neumann, H.: Ein Fall von Gelankerkrankung (Coxitis rechts) bei Filaria loa, Deutsche Ztschr. f. Chir. **227**:331 (Sept.) 1932.

10. Leger, M., and Queinnec, P.: Quelque mots sur un traitement de la filariose Loa, Bull. Soc. path. exot. **25**:690 (July) 1932.

result. They have found arsenicals and antimony to be ineffective. Intramuscular injections of a French proprietary medicine containing chloral and menthol gave complete relief in their case. Gougerot and Blum [11] claimed that a cure was obtained in one case by subcutaneous injections of sulpharsphenamine. Two series of treatments were given; the first consisted of doses of 0.4 Gm. each and the second of doses of 1.08 Gm. each.

In the present case we used fuadin, administered intramuscularly. It had no effect on the numbers of microfilariae found. The length of life of these parasites was, however, reduced from ten to two days when they were kept in an incubator at body temperature. The light from a carbon arc would cause the microfilariae to assume temporarily the stiff poker-like rigidity seen in the killed specimen. They would again resume their active motility when the light was removed.

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11. Gougerot and Blum, P.: Un cas de "filaria loa" guéri par des injections souscutanées de sulfarsénal, Bull. Soc. franç. de dermat. et syph. **39**:154 (Jan.) 1932.

CERVICAL SYMPATHECTOMY IN RETINITIS PIGMENTOSA

PRELIMINARY REPORT ON RESULTS

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Retinitis pigmentosa, starting in childhood and progressing gradually with or without stationary periods to more or less complete blindness, has not been strikingly influenced by any of the present methods of therapy. Any attempts, then, to halt the progress of the disease or ameliorate the existing disability should evoke considerable interest. In a brief report Royle, of Australia, presented five patients with retinitis pigmentosa in whom section of the sympathetic pathways to the eye was accomplished.[1] Two years later he reported on fourteen patients and presented visual fields showing definite increases following sympathectomy done early. Those with blindness of long standing did not respond.[2] In all these patients he used a method of section of the sympathetic trunk below the first thoracic ganglion.[3]

Favorable results, with increase in central and peripheral vision or improvement in night blindness, were reported by Meighen,[4] Campbell [5] and Magitot.[6] This group of authors removed the superior cervical ganglion, except Magitot, who stripped the carotid artery of its periarterial plexus. In this country, Holloway and Wheeler in a personal communication informed us of unreported cases. In Holloway's patients

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1. Royle, N. D.: The Treatment of Blindness, Associated with Retinitis Pigmentosa: A Preliminary Report, M. J. Australia **2**:364 (Sept. 13) 1930.

2. Royle, N. D.: Treatment of Blindness, Associated with Retinitis Pigmentosa, M. J. Australia **2**:111 (July 23) 1932.

3. Royle, N. D.: Sympathetic Trunk Section: A New Operation for Raynaud's Disease and Spastic Paralysis of the Upper Limb, M. J. Australia **2**:436 (Oct. 6) 1928.

4. Meighen, S. S.: Treatment of Retinitis Pigmentosa by Sympathectomy, Tr. Ophth. Soc. U. Kingdom **51**:124, 1931.

5. Campbell, G.: Sympathectomy in a Case of Retinitis Pigmentosa, Canad. M. A. J. **26**:674 (June) 1932.

6. Magitot, A.: La sympathectomie carotidienne comme thérapeutique de certaines affections dégénératives du nerf optique et de la rétine, Bull. Acad. de méd, Paris **111**:816 (June 12) 1934.

Frazier performed a cervicodorsal sympathectomy, whereas in Wheeler's patients Hanford performed a superior cervical ganglionectomy. Caeiro, Malbran and Balza [7] reported five cases but without any perimetric determinations. Quite recently MacDonald and McKenzie [8] published four cases, in two of which slight improvement was shown, which was more marked in the patient with the shortest history of retinitis pigmentosa. These authors used a cervicothoracic sympathectomy, but decided that excision of the superior cervical ganglion with stripping of the carotid sheath would be more desirable. We have come to the same conclusion and have carried out this procedure in the later cases.

We have asked ourselves in connection with this problem the following questions: 1. Is there a demonstrable effect on the visual fields or the visual acuity of patients affected by retinitis pigmentosa following sympathectomy? 2. Can the progress of the disease be halted by this operation?

SURGICAL METHODS

In order to denervate the eye from sympathetic fibers one can resort to one of three procedures: A section of the thoracic sympathetic trunk below the first thoracic ganglion [3] interrupts the sympathetic outflow from the thoracic cord to the ganglionated trunk. There is no known connection between the cervical cord and the cervical sympathetic chain. This is the operation that Royle described for Raynaud's disease and spastic paralysis of the upper limb. It is open to criticism for three reasons: 1. The trunk when merely cut across regenerates rapidly and the effect of the sympathectomy is only temporary. 2. It also denervates the upper extremity, which is not desirable except in certain rare instances when retinitis pigmentosa is associated with Raynaud's disease. 3. The preganglionic fibers to the eye pass out from the cord in the white ramus of the first thoracic segment and thus might escape division unless the stellate ganglion is removed.

The second operation, based on the criticisms just mentioned in regard to Royle's method, is the typical cervicothoracic sympathetic ganglionectomy as customarily performed for certain vasomotor phenomena of the upper extremities. This consists in the removal of the stellate (inferior cervical and first thoracic) and the second thoracic ganglion with the intervening trunk and the gray rami going to peripheral nerves and vessels. The operation may be performed through a posterior approach as evolved and modified by Adson,[9] which necessitates the removal of portions of the first and often the second rib on

7. Caeiro, Malbran and Balza: Rev. Asoc. méd. argent. **47**:3403, 1933.

8. MacDonald, A. E., and McKenzie, K. G.: Sympathectomy for Retinitis Pigmentosa, Arch. Ophth. **13**:362 (March) 1935.

9. Adson, A. W.: Changes in the Technique of Cervicothoracic Ganglionectomy and Trunk Resection, Am. J. Surg. **23**:287 (Feb.) 1934.

both sides. Or one can approach the area by the method of Gask [10] from the front, the principal points being the transsection of the scalenus anticus muscle, the division of Sibson's fascia and the retraction of the pleura with the subclavian artery. Both methods have their adherents. In our opinion, the anterior approach, in spite of the greater depth of the wound and the technical difficulties, offers a procedure which involves less damage to the tissues and gives perfect exposure.

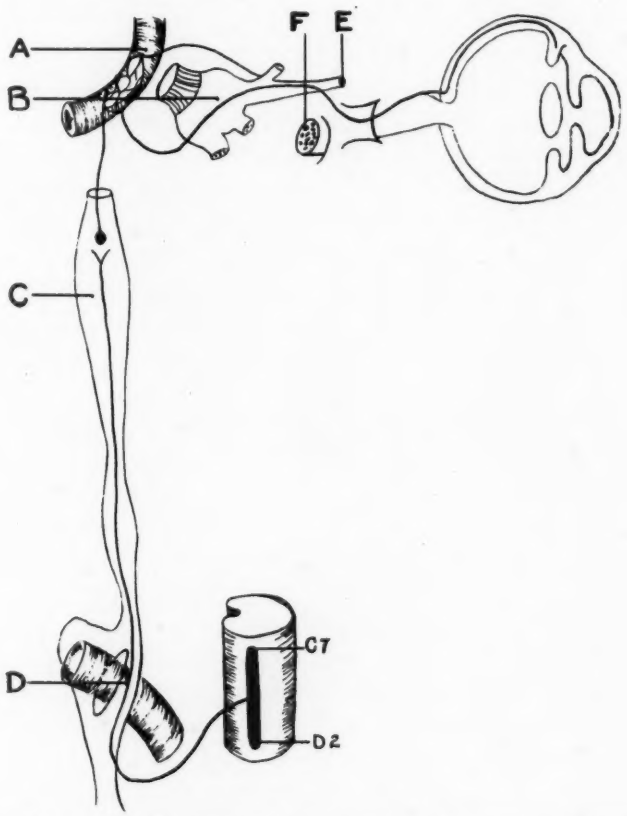

Fig. 1.—The principal sympathetic pathway to the eye: *A*, internal carotid artery; *B*, gasserian ganglion; *C*, superior cervical ganglion; *D*, ansa of Vieussens; *E*, nasociliary nerve; *F*, ophthalmic ganglion. From Delmas and Laux: Anatomie medico-chirurgicale du systeme nerveux vegetatif, Paris, Masson & Cie, 1933.

The criticism of this method is in part identical with the objection to Royle's operation, namely, that it denervates unnecessarily large areas. It also leaves intact the postganglionic fibers to the eye, which originate mostly in the superior cervical ganglion. There is some well

10. Gask, G. F.: Surgery of the Sympathetic Nervous System, Brit. J. Surg. **21**:113 (July) 1933.

grounded suspicion [11] that peripheral vasodilatation is maximal when the cells of the postganglionic fibers are removed, and cervicothoracic ganglionectomy leaves the synapsis at the superior cervical ganglion intact (fig. 1 *A*).

This leads us to the third possibility, namely, the excision of the superior cervical ganglion lying behind the neurovascular bundle in the upper part of the neck on the prevertebral fascia. We have approached it through an incision anterior to the sternocleidomastoid muscle. This operation destroys the excitor ganglion and the postganglionic fibers to the eye. And truly, following such an operation, one may observe a complete sympathetic paralysis of the iris, as the pupil fails to dilate with cocaine but is increasingly sensitive to epinephrine (cases 3, 4, 5 and 6). The only objection one might make to this method is that sympathetic fibers running along the carotid and vertebral vessels are thus left intact. They might serve as an auxiliary pathway of sympathetic impulses to the retinal vessels. To overcome this difficulty, one may add to the excision of the superior cervical ganglion a stripping of the internal carotid artery as advocated by Meighen,[4] whereas the insignificant sympathetic plexus of the vertebral artery can be ignored.

We have performed altogether eleven sympathectomies on six patients. Of these four were cervicodorsal and seven were superior cervical sympathectomies. The degree of sympathetic denervation deserves further analysis.

STUDIES TO EVALUATE THE DEGREE OF SYMPATHETIC DENERVATION

There is hardly any organ or region of the body which so readily lends itself to the study of the effects of sympathetic denervation as the eye. Aside from Horner's syndrome, which is typical when miosis, ptosis and enophthalmos are present, one can study the size of the vessels in the retina and determine the reactions of the pupil to cocaine and epinephrine.

Enophthalmos, according to the recent extensive investigation of Wagener,[12] is a very inconstant sign of cervical sympathetic paralysis, nor does it occur as an immediate result of cervicothoracic sympathectomy. A slight tendency to enophthalmos may be noted later, which is probably due to a shrinkage of tissues in the orbit. Only one of his ninety-four patients with Horner's syndrome had definitely demonstrable enophthalmos. Ptosis, if it once occurs, seems permanent and

11. Gask, G. E., and Ross, P. J.: The Surgery of the Sympathetic Nervous System, Baltimore, William Wood & Company, 1934.

12. Wagener, H. P.: Enophthalmos in Horner's Syndrome, Am. J. Ophth. **17**:209 (March) 1934.

does not improve with time. It is always incomplete, as only the smooth muscle levator is affected. When sympathectomy is unilateral it may be objectionable to the patient on cosmetic grounds. If Horner's syndrome is bilateral, however, it is not very noticeable. The contracted pupil which does not dilate under cocaine is the best proof of complete sympathetic denervation of the eye. Nevertheless, as observed by Wagener,[13] not all patients subjected to a typical cervicodorsal sympathectomy show failure of pupillary dilatation to cocaine. In our series a complete sympathetic denervation, as tested by the pupillary reaction to cocaine, was obtained in six of eleven operations. This finding emphasizes the difficulty of complete denervation and points to the excision of the superior cervical ganglion with stripping of the internal carotid artery as the simplest and optimal procedure.

The response of the pupil to epinephrine following sympathetic denervation is exaggerated. This confirms the general rule that the myoneural junction, when deprived of its sympathetic innervation, is increasingly sensitive to epinephrine. It is also true of peripheral vessels deprived of sympathetic influence.[14]

The permanency of vasodilatation following sympathectomy is a subject under considerable discussion. The immediate effect of sympathetic denervation is a marked dilatation of arterioles and capillaries. Within a few weeks, however, vascular tonus is regained, as first stated by Goltz and Freusberg[15] and demonstrated on man by Johnson, Scupham and Gilbert[16] using a small finger plethysmograph. This does not rule out, however, the possibility of a permanently increased supply of blood to the part. Herrick, Essex and Baldes[17] measured the velocity of the blood flow in sympathectomized animals with the Rein "Stromuhr" and found it to be increased several months after sympathectomy. Theis[18] showed, by cannulating the femoral artery below a ligature

13. Wagener, H. P.: Ocular Changes Following Cervicothoracic Sympathetic Ganglionectomy, S. Clin. North America **11**:867 (Aug.) 1931.

14. Freeman, N. E.; Smithvick, R. H., and White, J. L.: Adrenal Secretion in Man; Reactions of Blood Vessels of Human Extremity Sensitized by Sympathectomy to Adrenalin and to Adrenal Secretion Resulting from Insulin-Hypoglycemia, Am. J. Physiol. **107**:529 (March) 1934.

15. Goltz, F. R., and Freusberg, A.: Ueber Gefässerweiternde Nerven, Arch. f. d. ges. Physiol. **9**:174, 1874.

16. Johnson, C. A.; Scupham, G. H., and Gilbert, N. C.: Studies on Peripheral Vascular Phenomena: Observations on Peripheral Vascular Changes Following Unilateral Cervical Ganglionectomy and Ramisectomy, Surg., Gynec. & Obst. **55**:731 (Dec.) 1932.

17. Herrick, J. F.; Essex, H. A., and Baldes, E. J.: Effects of Lumbar Sympathectomy on the Flow of Blood in the Femoral Artery, Am. J. Physiol. **101**:213, 1932.

18. Theis, F. V.: Effect of Sympathetic Neurectomy on Collateral Arteriole Circulation of Extremities: Experimental Study, Surg., Gynec. & Obst. **57**:737 (Dec.) 1933.

in dogs, that following sympathectomy the blood volume flow per minute was permanently increased. One of us [19] has found an increase in the oxygen content of the venous blood draining the sympathectomized area. These findings make it seem that although the intrinsic vascular tonus of the sympathectomized vessel is regained there is some suggestive evidence of a permanently improved irrigation of the tissues with oxygenated blood.

The effect of sympathectomy on vascular permeability has been studied in only one patient. In this girl (case 4) 4 Gm. of fluorescein was ingested, and its appearance and intensity were noted in the anterior chambers of the sympathectomized and the control eye. Observations made at one-half, two, two and one-half and three hours showed a greater permeability (better diffusion) in the eye homolateral with the operation.

The effect of the sympathetic nervous system on the metabolism of pigment, on the migration, contraction and dilatation of melanotic pigment, is summarized by Parker.[20] In cases of retinitis pigmentosa, however, the migration of pigment is generally considered as secondary to a primary trophic or vascular degeneration of the retina. Of interest, too, is the possible relation of this disease to the pigment hormone of the intermediate part of the pituitary.[21] This hormone may very well act through the intermediary of a sympathetic impulse. Such a relationship has been shown to exist by Lucke between the diabetogenic principle (contrainsular hormone) of the anterior pituitary and the adrenal, which cannot be stimulated by it following sympathetic denervation.[22]

REPORT OF CASES

CASE 1.—Donald A., aged 18, a patient of Dr. Frank Brawley, had noticed night blindness for four years. The fundus picture on July 6, 1933 was considered typical of retinitis pigmentosa. Vision with correction was 6/12 in each eye, and the visual fields were within 20 degrees in each eye (fig. 2*A*). Sympathectomy on the left side was performed on October 2, but without production of Horner's syndrome. The visual fields were practically unchanged on November 14. On Jan. 7, 1934, right sympathectomy was performed with production of Horner's syndrome, typical on the right side. On February 23 vision with correction was: right eye, 6/10—1, left eye, 6/10—1. The visual fields on this date are shown in figure 2*B*. The left field was practically unchanged, while the

19. de Takats, G.: Unpublished data.

20. Parker, G. H.: Humoral Agents in Nervous Activity, London, Cambridge, University Press, 1932.

21. Zondek, B.: Chromatophorotropic Principle of the Pars Intermedia of the Pituitary, J. A. M. A. **104**:637 (Feb. 23) 1935. Jores, A.: Melanophorenhormon und Auge, Klin. Wchnschr. **12**:1599, 1933.

22. Wilder, R. M., and Wilbur, D. L.: Diseases of Metabolism and Nutrition, Arch. Int. Med. **55**:304 (Feb.) 1935.

right field showed the same outline for a 1 degree white test object but a slight increase in that for colors.

CASE 2.—Celia S., aged 33, a patient of Dr. Beulah Cushman, gave a history of night blindness for twenty-two years. Vision on Aug. 22, 1933 was 8/10 in each eye, and the visual fields for 5/330 white were within 10 degrees (fig. 3 *A*). The fundus picture was typical of retinitis pigmentosa. Both lenses had absorbed following previous discissions for cataracta complicata. On Feb. 7, 1934, cervical sympathectomy was performed on the right side, and on August 22 this was repeated at the patient's request on the left side. Following the first operation

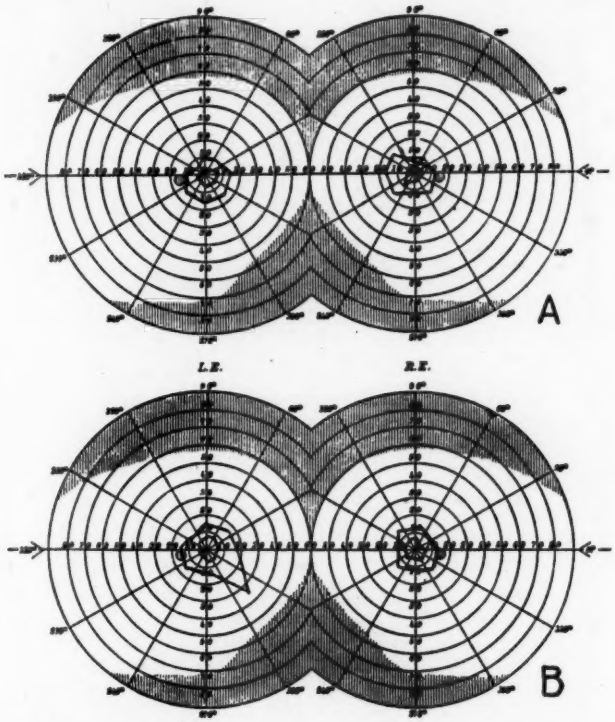

Fig. 2.—The visual fields in case 1: *A*, when first examined, on July 6, 1933 (1 degree white and red); *B*, on Feb. 23, 1934, five months after left and seven weeks after right sympathectomy (1 degree white and red).

the right disk appeared of better color (Dr. Cushman), and the patient believed that vision at night was considerably improved.

On November 26, vision was the same as at first: 8/10 with correction. The fields were also unchanged (fig. 3 B). Adaptation tests, which had not been made previous to the first operation, showed a marked reduction in light sense, but this was slightly better in the right eye (side of previous operation). The tests have not been repeated.

CASE 3.—Frank D., aged 27, showed the typical picture of retinitis pigmentosa. Vision had been poor since the age of 6. Vision with correction was: right, 18/200, left, 20/200. There was loss of the upper half of both visual fields with a large central scotoma.

Left cervical sympathectomy was performed on April 25, 1934. Horner's syndrome developed and was complete. Six months later, vision with correction was 8/200 in each eye. The fields were practically unchanged.

Case 4.—Lydia B., aged 32, had been under observation in the Northwestern University Eye Clinic since October 1932. She showed the fundus picture of retinitis pigmentosa, with marked contraction of the visual fields. She had received ten subcutaneous injections of a suspension of uveal pigment. During this treatment the fields and vision remained unchanged. Vision, right, was 20/40; left, 20/20—3 (fig. 4 *A*). One year later the field showed considerable further contraction (fig. 4 *B*). Vision with correction was: right, 20/40, left,

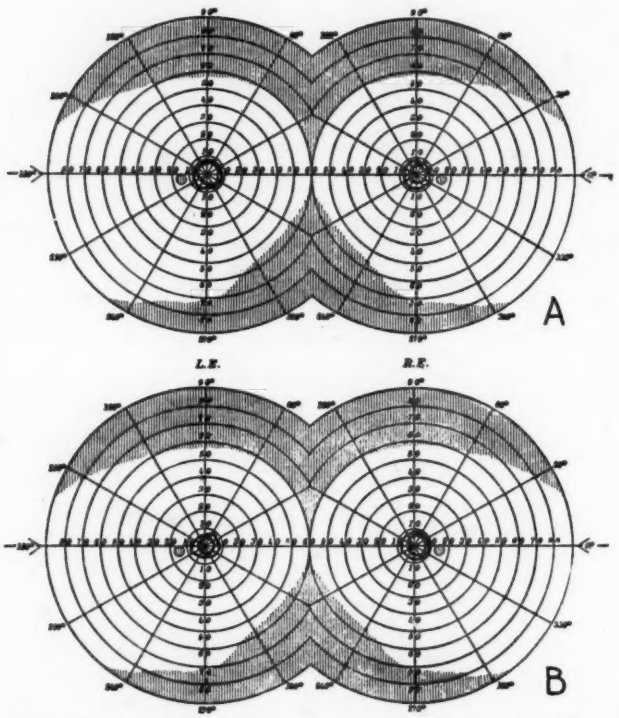

Fig. 3.—The visual fields in case 2: *A*, when first examined, on Aug. 22, 1933 (5/330 white and red); *B*, on Nov. 26, 1934, ten months after right and three months after left cervical sympathectomy (5/330 white and red).

20/20—3. On March 3, 1934, right cervical sympathectomy was performed. Three weeks later, Horner's syndrome was present on the right, well marked, with flushing of the conjunctival vessels of the right eye. No change in the vascularity of the fundus or in the color of the disks could be observed. Vision was recorded as slightly better (20/15—4) in the left eye (no operation on that side) but was unchanged in the right eye, and the fields showed no change. A test for permeability of the intra-ocular capillaries after injection of 4 Gm. of fluorescein showed greater permeability on the right side after one-half, two, two and one-half and three hours.

In May, the condition being unchanged, left cervical sympathectomy was performed at the patient's request. Horner's syndrome was not observed on the left

side following this operation. Vision remained the same in July, while the fields showed a slight increase, especially on the right side. Adaptation tests with Tscherning's photometric glasses in August showed a slightly better adaptation on the left side. In November, eight months after the first and five months after

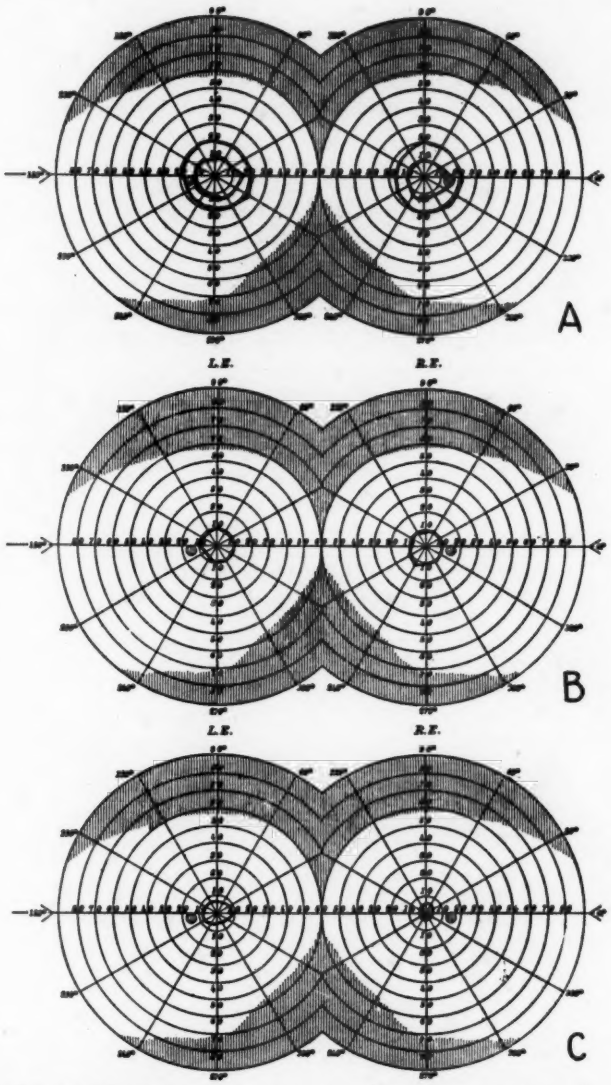

Fig. 4.—The visual fields in case 4: *A*, when first examined, in October 1932 (3/330 white and 6/330 red); *B*, one year later (3/330 white); *C*, in November 1934, eight months after right and five months after left cervical sympathectomy (3/330 white).

the second operation, vision was unchanged: right, 20/40 +2; left, 20/15 —2. The fields, however, showed contraction further than that present at the time of the first operation, and more on the left than on the right side (fig. 4 *C*). On the left side, however, no complete sympathetic denervation was accomplished.

CASE 5.—Bertha S., aged 15, showed the fundus picture of retinitis pigmentosa. Vision with correction was: right, 20/25, left, 20/40 +1. The visual fields for 3/330 white are shown in figure 5 *A*.

Right cervical sympathectomy was performed on Jan. 16, 1934, but without production of Horner's syndrome. No changes in the fundi were observed. On July 14, left cervical sympathectomy was performed, resulting in Horner's syndrome on that side, which was complete. No change in the fundus vessels was noted and in November, five months after the second operation, vision was: right, 20/40 —1, left, 20/40 —1. The fields were unchanged (fig. 5 *A* and *B*).

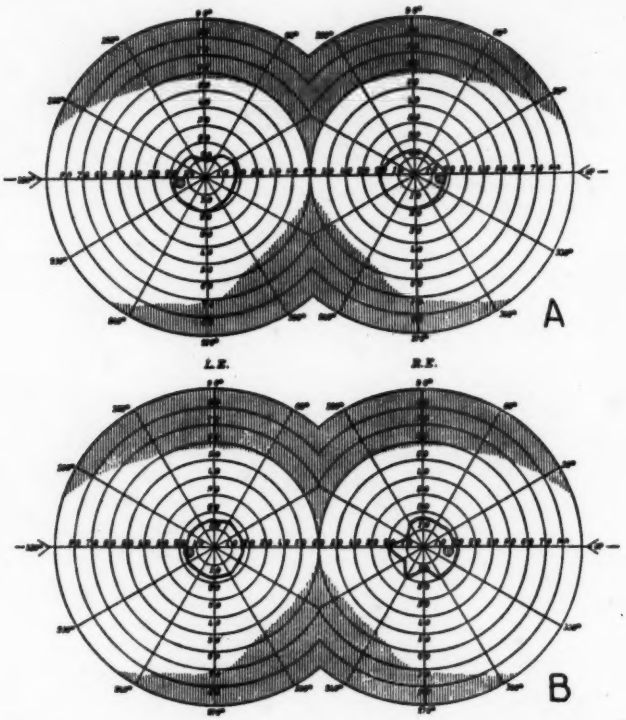

Fig. 5.—The visual fields in case 5: *A*, when the patient was first seen (3/330 white); *B*, eleven months after right and five months after left cervical sympathectomy (3/330 white).

CASE 6.—Jens S., a man of 50, showed the picture of retinitis pigmentosa, though he had noted poor vision for only two years. With the right eye he was able to count fingers at 2 feet (61 cm.). With the left eye vision with correction was 20/50. Cervical sympathectomy was performed on the left side on Sept. 26, 1933, resulting in Horner's syndrome, incomplete. This operation was repeated on the right side on March 7, 1934; Horner's syndrome resulted and was complete. Two months later vision and the visual fields remained unchanged. The retinal vessels on the right side were considered somewhat larger than those on the left, but were quite small on both sides.

COMMENT

From the pathologic and ophthalmoscopic picture of retinitis pigmentosa it is obvious that patients with the late condition, with evidence

of massive bone corpuscles and corresponding impairment of visual fields, could hardly be benefited unless by preservation of the preexisting vision. In patients with the earlier condition an improvement in night blindness with perhaps an increase of visual acuity might be hoped for. In not a single instance have we observed an enlargement of visual fields or an increase in visual acuity as stated in previous reports.[23] Of our six patients, three gave a history of night blindness or poor vision for twenty-two, twenty-three and thirty years, respectively. They have not been benefited, nor has their disease progressed, but our first operation was performed on Sept. 26, 1933, and a year and a half is too short a time to permit an observation on the progress of this disease. Of the remaining three patients, one (case 1) has not shown complete sympathetic denervation of either eye. He should be readmitted shortly for operation. One (case 4), while showing no increase in the visual fields and acuity, believes her night blindness has improved. Tests for dark adaptation and minimal light perception were not made before the operation. A young girl of 15 (case 5), with Horner's syndrome complete on one side and incomplete on the other, notes no benefit even in regard to night blindness. No constant changes in vascularity of the fundus were noted, though increased congestion of the conjunctival vessels was always present on the side of Horner's syndrome and persisted for several months or longer. The judgment of slight changes in the state of the retinal vessels is a difficult one to make, but in no case were the changes striking.

It is our impression, then, based on observation of six patients subjected to eleven operations, that the enthusiastic reports on increase in the visual fields and visual acuity could not be substantiated. Our longest observation is a year and a half, the shortest eight months. In only one of the patients with the condition in an early stage was there a subjective improvement in night blindness, but since we have no preoperative observations this statement cannot be subjected to objective control. The lead is important enough, however, to suggest further observations on future patients in regard to dark adaptation and minimal light perception. Whether the operation is capable of arresting the disease cannot be stated before several years have elapsed.

A word of caution must be sounded in regard to Magitot's procedure. He claimed a great increase in retinal vascularity, visual acuity and visual fields following the stripping of the common carotid artery, its bifurcation and the bulb of the internal carotid artery and the removal of the carotid gland. He claimed a permanent retinal hypertension and the best results in toxic neuroretinitis with constriction of vessels.

23. Royle.[1] Royle.[2] Meighen.[4] Campbell.[5] Magitot.[6] Caeiro, Malbran and McKenzie.[7]

The procedure he described has been known to physiologists since Hering's important contribution as "denervation of the carotid sinus" and deprives the body of a very important pressoreceptor mechanism for the control and stabilization of the blood pressure.[24] In patients with increased blood pressure it may elevate and stabilize blood pressure at a high level. One of Magitot's patients remained dizzy and suffered occipital headaches for months after the operation.

The operation constitutes such a serious interference with the normal pressoregulator mechanism that its use until further study cannot be recommended. Its effect depends on raising general and intracranial blood pressure.

SUMMARY

A sympathetic denervation of the eye was attempted to improve the visual acuity and visual fields of six patients suffering from retinitis pigmentosa.

Of eleven operations, four consisted of cervicodorsal and seven of superior cervical ganglionectomies. The latter, particularly if combined with a stripping of the internal carotid artery, seems to be the simplest and most constantly reliable method of obtaining complete sympathetic denervation.

The test of complete sympathetic denervation was the failure of cocaine to dilate the contracted pupil. Not one of these patients showed an increase in visual acuity or in the visual fields. One younger patient reported an improvement in night blindness. None of them showed progress of the disease, but the longest period of observation was only a year and a half.

Further studies seem indicated on the effect of sympathectomy on dark adaptation and minimal light perception in these patients. Judged by our present methods, the only possible benefit is that which might arise from halting the progress of this disease.

A later report will be necessary to recommend or condemn this procedure definitely.

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24. See literature cited by Koch, E.: Die reflektorische Selbststeuerung des Kreislaufes, in Kisch, B.: Ergebnisse der Kreislaufforschung, Dresden, Theodor Steinkopff, 1931, vol. 1.

A CHECKING STATION FOR TONOMETERS

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NEW YORK

Whenever one uses the Schiötz tonometer, one inevitably asks oneself the question: "Is my reading reliable?" It being assumed that the technic used is correct, there still remain two factors to be considered: the eyeball and the instrument. Since the tonometer measures merely the degree of impressibility of the cornea, the readings will vary slightly with such factors as the rigidity and thickness of the cornea and the size and shape of the eyeball. However, these individual variations are of minor importance and, at any rate, are unavoidable.

The accuracy of the tonometer itself plays decidedly the most important rôle in determining the reliability of the readings. Since the tonometric deflections have to be translated into millimeters of mercury by referrence to Schiötz' graphs, it is, of course, essential that the tonometer one uses should be an exact replica of the original one with which the graphs were obtained. That this is far from being the case is borne out by the observations of such authorities as Schiötz, Arnold and Comberg, all of whom found a striking lack of uniformity among tonometers. This is due partly to prolonged or improper use, but chiefly to faulty construction.

Checking stations for Schiötz tonometers were established in Tübingen (Arnold,[1] 1923), in Oslo (Schiötz,[2] 1925) and in Berlin (Comberg,[3] 1928). Curiously, no such facilities had been provided in the United States until the present time. At the suggestion of Dr. Mark J. Schoenberg and with the aid of the Grout Memorial Fund, a checking station was recently established at the Herman Knapp Memorial Eye Hospital, and it is hoped that ophthalmologists will avail themselves of its services.

The following factors influence the accuracy of a tonometer:

1. The weight of (*a*) the plunger mechanism, that is, the plunger, lever and 5.5 Gm. weight, and (*b*) the entire tonometer.
2. The radius of curvature of the foot-plate and of the plunger.
3. The diameter of the foot-plate and of the plunger.
4. The ratio of magnification, which depends on the length of the short arm of the lever and of the long arm of the lever.

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1. Arnold, H., and Karpow, C.: Ueber die Eichungsverfahren für das Tonometer von Schiötz, Klin. Monatsbl. f. Augenh. **71**:603, 1923.
2. Schiötz, H.: Tonometry, Brit. J. Ophth. **9**:145, 1925.
3. Comberg, W.: Ueber die Kontrolle des Schiötz Tonometers, Klin. Monatsbl. f. Augenh. **81**:289, 1928.

5. The accuracy of adjustment on the metal cornea.

6. The frictional resistance of the instrument.

The standard measurements of the parts referred to are indicated in the diagram. The weight of the plunger mechanism is determined by gradually lowering the tonometer by means of a lever to the pan of a beam balance until the pointer of the tonometer begins to move. By still further lowering the tonometer until the foot-plate rests on the balance the weight of the entire instrument is determined. The weight of the plunger should be 5.5 Gm. and that of the entire tonometer, 16 Gm. That the weight of the plunger is important goes without saying. The weight of the entire tonometer is also important, since

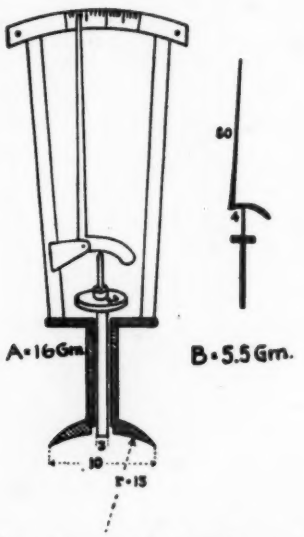

Fig. 1.—Diagram showing the essential parts of the Schiötz tonometer and their standard dimensions (numbers represent millimeters). *A* represents the part of the tonometer resting on the cornea, i. e., the tonometer with the 5.5 Gm. weight without the handle; *B*, the part which produces the impression in the cornea, consisting of the plunger, 5.5 Gm. weight, lever and pointer.

it compresses the contents of the eyeball and thus alters the intra-ocular pressure. The radius of curvature of the foot-plate should be 15 mm. The lower surface of the plunger should be concave and its radius of curvature also 15 mm. The diameters of the foot-plate and plunger are 10 and 3 mm., respectively. The long arm of the lever, represented by the pointer, should measure 80 mm., and the short arm, 4 mm., which gives a ratio of magnification of 1:20. The short arm is represented by the distance between the fulcrum and the tip of the plunger. As these two points lie in different planes, I have found it practical to measure their distance with the Wessely keratometer. Every tonometer possesses a certain amount of frictional resistance. To eliminate

that part of the resistance caused by the capillary attraction of tears in the space between the plunger and the barrel, the lower portion of this space has been widened into a gutter surrounding the plunger.

The tonometer used as the standard of the station with which I am associated was made and tested by John Schiötz, a son of the inventor. It was imported and is being used solely for the purpose of checking tonometers.

Each tonometer to be checked is compared with this master instrument with regard to the structural features previously enumerated. Then its readings are checked with those of the standard by placing the two successively on a rubber drum stretched across the top of a small metal cylinder which communicates with a water manometer. The set-up

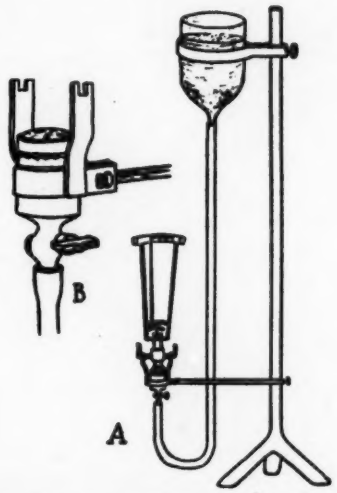

Fig. 2.—*A* shows the water manometer set up for comparing Schiötz tonometers with the master instrument; *B*, details of the metal capsule which is equipped with a stopcock and two arms for supporting the tonometer. The testing membrane which covers it is made from the finger-tip of a rubber glove.

is shown in the diagram. The water manometer serves merely as a convenient means of varying the pressure within the metal cylinder. The level of the manometer is so adjusted that the standard tonometer, when placed on the rubber drum, will give a deflection of 3 mm. with a 5.5 Gm. weight. This reading corresponds to 30 mm. of mercury on the new Schiötz graphs and marks the dividing line between normal and abnormal ocular tension. With this adjustment the 7.5 Gm. weight gives a reading of about 5 mm., and the 10 Gm. weight about 7 mm. The manometer can be used with the stopcock either open or closed; the first method has been adopted as the more convenient.

The crucial test of the accuracy of the tonometer is, after all, its performance on a patient's eyeball. I therefore tested several tonometers

both on the rubber drum and on patients, choosing for this purpose eyes with borderline tensions. The results obtained showed uniform correspondence between the two methods of checking.

In a series of thirty tonometers thus far examined, I found that twenty-one were accurate and nine inaccurate. These results should serve to emphasize still more the lack of uniformity among tonometers of various ages and manufactures and to point out the necessity of having one's tonometer periodically checked.

DISCUSSION

DR. WILLIAM MCLEAN: That a checking station for Schiötz tonometers is needed is shown by the fact that Dr. Posner has found that approximately one third of the Schiötz tonometers he has tested were inaccurate.

I cannot see that Dr. Posner has the right to assume that his tonometer is correct until he has checked it with a manometer in a manner that leaves no doubt. Schiötz was positive that the readings obtained with his models previous to that of 1926 were in accordance with those of the manometer, but the insistence by Priestley Smith that the Schiötz values were too low and the report of my findings in which I proved them too low caused Schiötz to undertake new investigations, the results of which led him to repudiate his findings prior to 1926 and to bring out a new tonometer with a new set of values that were higher than those concerning the corrections of which he was just as positive before. I believe that had Schiötz checked the findings which he made with the eyes of dead subjects, whereby he raised his set of values about 20 per cent, with standard readings of manometer pressures for living human eyes as I did, using an air bubble as a checking guide, he would have raised his values a little higher and they would then be in accordance with those indicated by my tonometer.

Dr. Posner states that he has found inaccuracies caused by the prolonged or improper use of tonometers. I should like to ask Dr. Posner when his tonometer gets out of order from prolonged use, by whom and how will it be checked? His tonometer is just as liable to get out of order from prolonged use as is any other, and he will not be aware that he has condemned by the inaccuracy of his own tonometer a tonometer given him for testing. One should check tonometers against a definite manometer reading and in a way that will not introduce errors.

Dr. Schoenberg invited me to a demonstration of this checking apparatus. Dr. Posner placed his tonometer with its 7.5 Gm. weight on the rubber cornea with the closed manometer, and the indicating needle registered 3 mm. on the scale. He then, without touching the tonometer, switched to the open method; the indicating needle should have receded to about 5 mm., but it did not move. The pressure within the cornea changed. Why did not the tonometer register the change?

This evening Dr. Posner has given a demonstration of his checking system for Schiötz tonometers. As I stated, Dr. Schoenberg invited me to the Herman Knapp Memorial Hospital, where I witnessed a demonstration. It seemed to me that at both these demonstrations the manometer registered a higher pressure than the equivalent of 30 mm. of mercury. Tonight after Dr. Posner had adjusted his tonometer, the instrument by which others are to be judged, so that its indicating needle with the 5.5 Gm. weight registered 3 mm. on the scale (which by the Schiötz graph is 30 mm. of mercury) the manometer actually registered 52 mm. of mercury. This discrepancy was called to his attention, and he made another adjust-

ment. The second adjustment required the manometer to be at 48 mm. of mercury in order that the Schiötz tonometer should indicate 30 mm. of mercury. Thus there was an actual difference of 18 mm. of mercury between the reading on the manometer and that on the Schiötz tonometer.

This is Dr. Posner's testing apparatus which he intends shall be a checking station for Schiötz tonometers, and it bears out my contention that the Schiötz tonometer still underestimates the true intra-ocular pressure (author's abstract).

Dr. Adolph Posner: I wish to thank Dr. McLean for having brought out some interesting features which I intentionally did not mention. His discussion of his own work, though interesting, has no bearing on the subject at hand. In reply to his criticism of my work, I can only repeat that its purpose is not to set a new standard for tonometers; neither is its purpose to calibrate the Schiötz tonometer. The aim of the checking station is merely to help the ophthalmologist, who either has a very old and badly used tonometer or buys a tonometer of an unknown make for a fraction of the price charged for the original tonometer, with no guarantee as to accuracy, to know whether his tonometer measures up to the original Schiötz model which I am using as the master instrument.

I am fully aware of the inherent defects of the rubber membrane in simulating the human eyeball, but since my method consists in the simultaneous and relative comparison of two tonometers of the same construction these defects do not vitiate the results. Dr. McLean has confined himself to a condemnation of the rubber membrane, overlooking entirely the fact that I have given it secondary importance, as I place much more emphasis on the exactness of the construction of the tonometer.

Dr. McLean's contention that my own tonometer will also grow old is too theoretical to be taken seriously. Age per se does not impair the accuracy of an instrument. As I have emphasized before, my tonometer is being used solely for checking and thus is subjected neither to careless handling nor to the corrosive action of tears. Moreover, Dr. McLean knows very well how much care Professor Schiötz gave to the choice of the metal used for the pointer and to the construction of the entire instrument, so as to reduce to a minimum the warping caused by changes in climate and temperature.

And, finally, if tonometers get out of order as easily as Dr. McLean claims they do, what does he do about checking his own tonometers from time to time?

Dr. Mark J. Schoenberg: The fundamental question regarding the accuracy of the tonometer is to determine whether the weight of the tonometer and of its various component parts as well as the proper construction of the instrument exactly reproduce those of the Schiötz tonometer.

RETINOSCOPY AT A DEFINITE DISTANCE

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The art of retinoscopy is based theoretically and practically on the maintenance of a known distance between the eye of the patient and that of the retinoscopist. The length actually chosen is based on habit and convenience, and may be, and frequently is, modified to suit unusual circumstances. The dioptric effect of this distance is, of course, well known: Compensation is made (for infinity) by subtracting from the retinoscopic findings the number of diopters which is the reciprocal of the distance in meters. The observer places before the eye of the patient a lens which will cause the emergent light to come to a focus at some convenient and accurately measured distance. The reciprocal of this focal length (in meters) yields the dioptric value of the lens which causes parallel light to be focused at that specific distance; and this dioptric value subtracted from the lens actually in place yields the lens which renders the emergent light from the fundus parallel, and renders the patient emmetropic. For example: if the focus is observed at 1 meter, 1 diopter would be subtracted; if the focus is observed at 0.5 meter, 2 diopters would be subtracted, and so on.

At what distance should the emergent light be observed? Distances of several meters are impracticable. Distances much greater than 1 meter are quite inconvenient for making changes in the lenses in the refracting unit and even more inconvenient for making these changes in a trial frame. A stool on readily movable casters is very convenient at any distance. Also, since the intensity of illumination drops off with increasing distance, since it varies as the reciprocal of the square of the distance, intensity of illumination becomes poorer. As will be seen, from the standpoint of lessening the effect of errors in distance, the larger distances are preferable.

If x is the focal distance in meters, and if y is the number of diopters (to be subtracted to render the light parallel), the relation is $y = \frac{1}{x}$, by definition, and from the differential calculus is obtained the derivative, $\frac{dy}{dx} = -\frac{1}{x^2}$, or approximately $\Delta y = -\frac{\Delta x}{x^2}$ in the incremental notation.

The graphs of the original function, $y = \frac{1}{x}$, and of the derived function, $\frac{dy}{dx} = -\frac{1}{x^2}$, show many significant relationships nicely. The

Read before the Luther C. Peter Eye Clinic of the Graduate Hospital of the University of Pennsylvania, Philadelphia, at the meeting in January 1933.

graph of the function $y = \frac{1}{x}$ is a rectangular hyperbola, whose branches have the coordinate axes as asymptotes. For positive values of x, it is the branch shown in chart 1. On this graph it is to be noted that at the small distances there are reciprocally large dioptric effects, and that at the large distances there are reciprocally small dioptric effects.

The graph of the derived function, $\frac{dy}{dx} = -\frac{1}{x^2}$, is also a two-branched curve of hyperbolic type. For positive values of x, it is the

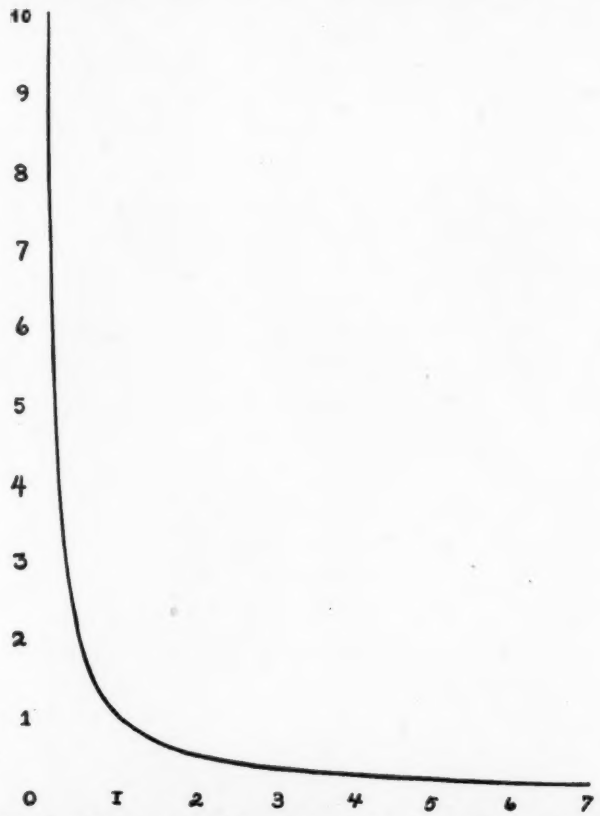

Chart 1.—The numbers at the left represent dioptric values; those at the bottom, distance in meters. The relationship between diopters (y) and focal distance (x in meters) is $y = \frac{1}{x}$. At small distances there are reciprocally large dioptric effects; at large distances there are reciprocally small dioptric effects.

branch shown in chart 2. This curve demonstrates the rate, $\frac{dy}{dx} = -\frac{1}{x^2}$, at which the dioptric value changes as the length changes, at different distances. It is to be noted that at the large distances the dioptric values change very slowly, the change being small even for tremendous changes in distance; at the short distances, particularly those under 1 meter, the dioptric values change very rapidly, undergoing enormous changes for small changes in distances. For example: at 5 meters the dioptric

value changes one twenty-fifth as fast as the distance, while at 0.5 meter the dioptric value changes four times as fast as the distance, and each time in a contrary sense, as indicated by the negative sign. Since the rate of change is great at short distances, small errors in distance are conducive to large dioptric errors. This is more obvious from the incremental form of the equation, $\Delta y = -\frac{\Delta x}{x^2}$ (approximately), in which Δy represents the change in y (diopters) induced by Δx, a

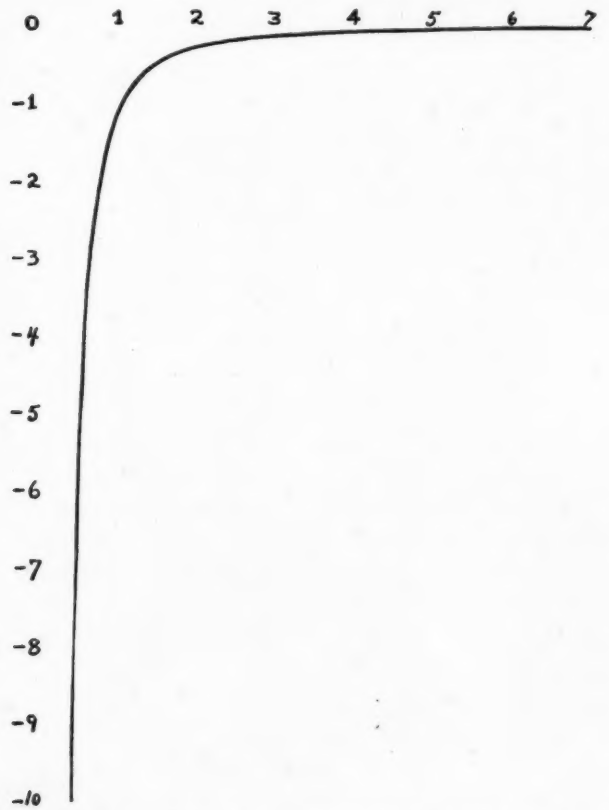

Chart 2.—The numbers at the left represent the rate ($\frac{dy}{dx}$) at which the diopters change as the distance ($\times$) changes, at different distances represented by the numbers at the top. The relationship is $\frac{dy}{dx} = -\frac{1}{x^2}$. At large distances the dioptric values change very slowly as distance changes; at small distances the dioptric values change very rapidly as distance changes. The negative values of the rate of change ($\frac{dy}{dx}$) indicate that the dioptric values decrease with increasing distance, and conversely.

small change or error in x (meters). It is seen again from this equation that small errors in x are relatively inconsequential at large distances; and that similar small errors in x are of great importance at

short distances. Or, the smaller the value of x, the greater are the incremental errors produced in y by decremental errors in x, and vice versa.

A further examination of the graph of the derived function shows that for practical purposes, at increasing distances greater than 1 meter, this liability to large dioptric errors is moderate to insignificant. But it is precisely these larger distances that are otherwise impracticable for routine retinoscopic examination. For decreasing distances of less than 1 meter this liability to large dioptric errors is moderate to enormous. At distances under 0.5 meter the diopters change more than four times as fast as the distance changes, and at an ever increasing rate the shorter the distance, so that these short distances are inadvisable for ordinary purposes.

At 0.33 meter the diopters are changing nine times as fast as the distance and in the opposite sense. An error of —1.33 cm. or +1.45 cm. in the distance presumed to be 0.33 meter would result in an error of ±0.12 diopter; similarly an error of —2.58 cm. or +3.03 cm. would result in an error of ±0.25 diopter. Nevertheless, this length is used in dynamic retinoscopy to ascertain the metropic state for the reading distance.

For usual purposes one may arbitrarily limit the viewing distance to the practicable range from 0.5 to 1.33 meters, since there is no distance mathematically optimal.[1] Several modes of operation are possible. One may choose a fixed and accurately maintained distance whose dioptric effect will be known from the reciprocal relationship between diopters and meters, and by changing the lens in the trial frame arrive at one which causes the light emergent from the fundus to focus at the selected distance. The dioptric value dependent on this distance is, of course, subtracted. This is the commonly practiced method and may be termed retinoscopy at a fixed distance. The distance usually chosen is 1 meter, principally because it renders the mathematics of subtraction quite easy. To stabilize the distance for retinoscopy at fixed distance,

1. The second derivative, $\frac{d^2v}{dx^2} = \frac{2}{x^3}$, shows the rate of change in $\frac{dv}{dx} = -\frac{1}{x^2}$. This is in turn the rate of change in the initial function, $y = \frac{1}{x}$, so that the second derivative is the rate at which the rate itself changes. This is 1 when $x = \sqrt[3]{2}$, 1.26 (approximately); so that at distances greater than this the rate of change is arithmetically small and is itself changing slowly, while at distances less than this the rate of change is arithmetically moderate to large and is itself changing rapidly. The distance 1.26 meters is significant for this reason (1.33 meters is actually chosen because it represents 0.75 diopters, the other representing a difference from this of less than 0.12 diopters). The actual dioptric change involved in increasing the distance beyond this point is so small as to make increases in distance beyond this impracticable in the method of retinoscopy at variable distances.

a cord may be used.[2] Desiring a 1 meter distance, the senior office associate [3] has for many years made use of a 1 meter cord, one end of which is permanently attached to the retinoscope handle, and the other end of which is provided with a hook or loop for attachment to the refracting unit or trial frame. This results very satisfactorily and effects refractions by skiascopy which always check accurately with the postretinoscopic verifications by subjective tests while the patients are still under cycloplegia.

Another mode of operation is possible. Within the range of distances chosen, the eye of the observer may be moved in reference to the eye of the patient, seeking the exact distance at which the focus is located, i. e., the point of reversal. If it is not within this range of distances, the lens in the trial frame is altered in an effort to bring it within this range. An example will make this clear: With divergent light from the retinoscope, if the latter is at 1 meter's length, in a particular meridian the reflex moves with using a + 3.00 diopter lens (which signifies that the emergent light has not come to a focus within the distance of observation) and moves against using a + 4.00 diopter lens (which signifies that the light has already come to a focus within the distance of observation); by shortening the length one can accurately find the focal distance and by subtracting the corresponding dioptric value determine the lens which renders the eye emmetropic in that meridian. This may be termed retinoscopy at variable distance, and to facilitate this the cord is marked into intervals representing diopters, i. e., reciprocals of distance. Working within the distance range 0.5 to 1 meter, the cord is divided by beads, knots or other marking devices into lengths whose reciprocals represent 1.00, 1.12, 1.25, 1.50, 1.75 and 2.00 diopters: the distances being 1.00, 0.89, 0.80, 0.67, 0.57 and 0.50 meter, respectively, from the trial frame. This yields a range of 1 diopter for further decreasing *algebraically* the net value of the lens before the eye, as corrected for infinity, beyond the correction applied for the 1 meter distance.[4]

This procedure of finding the point of reversal within a particular range of distances with few lens changes, instead of causing the focus to come to a specified unaltered distance by repeated lens changes, makes for more rapid and more accurate retinoscopy. It is more than a refine-

2. Goldnamer, W. W.: Meter Measure for Retinoscopy, Am. J. Ophth. **6**:685 (Aug.) 1923.

3. Dr. Ira A. Abrahamson, the senior office associate, initiated me into the art of retinoscopy and taught me the use of the 1 meter cord.

4. A 1.33 meter cord with these same divisions yields a diopter range from 0.75 to 2.00 diopters, a range of 1.25 so that working from an initial 1 meter distance the net value of the lens before the eye as corrected for infinity may be further decreased algebraically 1.00 diopter, or increased algebraically 0.25 diopter, over the correction applied for the 1 meter distance.

ment—it is almost a necessity—in the higher ranges of refractive errors, where the trial case lenses are not supplied in dioptric values sufficiently close to one another, although this criticism is not so applicable when a refracting unit is used.

There is yet one other consideration. At times a 1 meter distance is inconvenient for seeing the reflex. This is the condition frequently when there are marked axial or periaxial opacities in the cornea or lens or both. The reflex may then be observed more readily by decreasing the distance of observation. This difficulty is sometimes encountered also when there are small pupils, either because they are undilated (e. g., owing to contraindication to the use of a mydriatic-cycloplegic) or because they are undilatable (e. g., owing to iridolental synechiae). The graded intervals are essential if accurate metropic results are to be obtained at the reduced distances, and of course the longest utilizable distance yields the best results.

SUMMARY

1. The importance of accuracy in distance in minimizing dioptric error is presented.

2. The advantages and disadvantages of the various distances are discussed.

3. A practicable metric method is described for maintaining a fixed length between the patient and the observer for retinoscopy at a fixed distance.

4. A method is presented for performing retinoscopy at variable distances within a fixed range, with the following corollary:

5. The means is at the same time presented for accurately shortening the customary working distance (or range of distances) so that the reflex may be seen with greater facility under conditions in which it is obscured.

IS FULL CORRECTION OF VALUE IN CHECKING THE PROGRESS OF MYOPIA?

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NEW YORK

The question of the importance of the effect of full correction on the progress of myopia has been disturbing my peace of mind for a number of years. It is only natural that during the years spent at the ophthalmic clinic at the University of Bern I devoted part of the time to the study of the problem. The enormous amount of material, consisting of 20,188 clinical patients, was made available through the courtesy of Professor Siegrist, director of the clinic. The results of this work are submitted in the hope of bringing some solution to the question as yet unsolved.

A five year period, from 1916 to 1920, was selected, and the 2,166 persons with myopia, or 10.5 per cent of all the patients at the clinic, were studied. The study of the problem included: (1) an estimate of the average number of times a patient came to the clinic during the period studied; (2) a consideration of whether the myopia was stationary or progressive, only those persons being considered who were wearing full correction; (3) a consideration of the frequency of complications in the patients with correction and (4) a study of the other important factors besides near work and accommodation which influence the progress of myopia.

Of a series of 2,166 myopic patients, 772 were chosen because each of them came to the clinic not less than three consecutive times during the period studied. All the important conclusions were drawn from the study of these patients.

To be sure that these patients varied little from the usual type of patient seeking medical aid at the free clinic, the total number of patients seen at the clinic during the five year period were studied in relation to the number of patients with myopia.

The number of patients with myopia did not vary greatly for each year, ranging between 9 and 13.7 per cent of the total number of patients, the average being 10.5 per cent. Of these, 877, or 40.4 per cent, were men, and 1,289, or 59.6 per cent, were women.

Detailed study of the myopic patients according to age and sex reveals that there is a similar and characteristic rise in the number of patients of both sexes at the period of adolescence (from 10 to 30 years), as is shown graphically in chart 1.

In contrast to this, table 2 shows that the greatest number of cases of high myopia with complications occurs after the period of adolescence, the increased incidence beginning with the age of 30 and gradually rising to reach the highest point at the age of 50.

TABLE 1.—*Comparison of Total Number of Patients with Number of Those with Myopia*

Year	Total No. of Patients	Patients with Myopia	Percentage
1916	4,064	560	13.7
1917	4,112	383	9.0
1918	3,974	389	9.5
1919	4,006	417	10.4
1920	4,032	417	10.3
Total	20,188	2,166	10.5

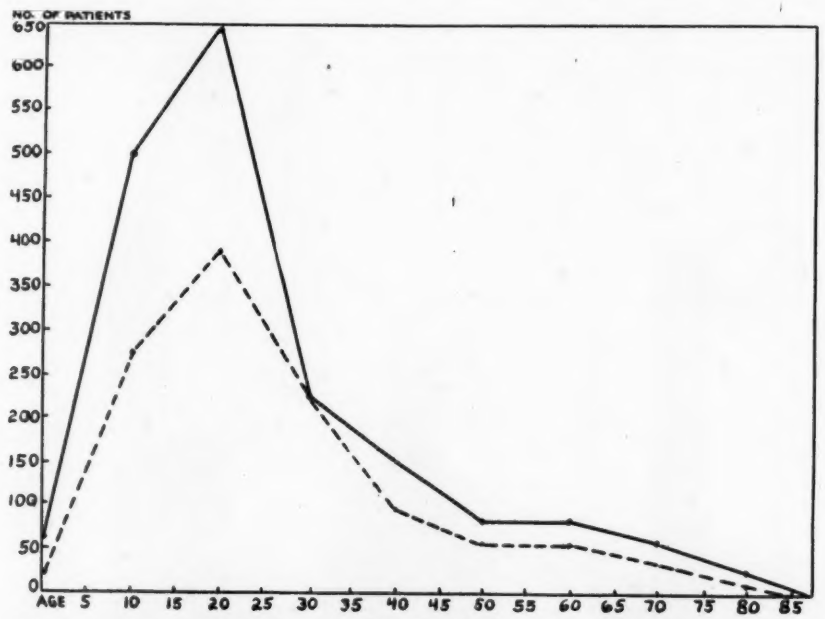

Chart 1.—Curves showing the similarity in the rise and fall of the number of myopic patients of both sexes at corresponding ages. The solid line is the curve for the women and the broken line, for the men.

Next the group of 772 patients on whom a follow-up study was made was divided into those in whom the disease had a tendency to progress mildly and those with so-called "malignant or rapidly progressing myopia," in which uninterrupted progression with all its complications is manifested at an early age. The first group consisted of 345 patients, or 44.6 per cent, and the second group, of 220 patients, or 28.4 per cent. Altogether, 73 per cent of all the cases in which a

follow-up study was made showed a tendency to progress despite the fact that the patients wore full correction at all times.

In line with this it was logical to study the effect of malignant myopia on the acuity of vision. Here a study of chart 2 is most useful.

TABLE 2.—*Incidence of Cases of High Myopia According to Age and Sex*

	Male			Female		
Age	Total No. of Cases of Myopia	High Myopia	Percentage	Total No. of Cases of Myopia	High Myopia	Percentage
Up to 20	393	76	19.3	649	114	17.5
20 to 30	227	29	12.7	227	57	25.1
30 to 40	91	20	21.9	152	49	32.2
40 to 50	66	20	30.3	99	38	38.3
50 to 60	62	18	29.3	99	31	31.4
60 to 70 and over	38	7	18.4	63	13	20.6
Total	877	170	19.3	1,289	292	22.6

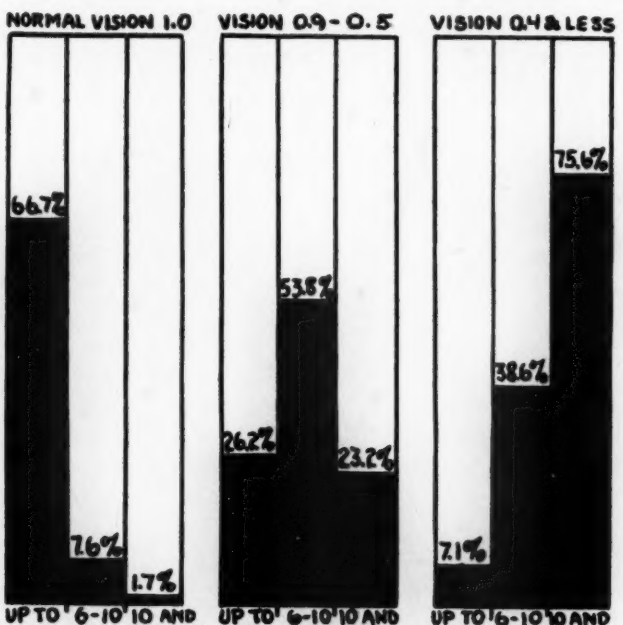

Chart 2.—Effect of malignant myopia on the acuity of vision. Of the patients with correction not exceeding 6 diopters, 66.7 per cent have normal vision and 7.1 per cent have less than half the normal vision. Of the patients wearing correction of 10 and more diopters, only 1.7 per cent have normal vision and 75.6 per cent have less than half the normal vision.

The patients studied did not include those with abnormal changes other than the direct outcome of myopia. From a total of 2,166 patients, 3,868 eyes were accepted as qualifying the demands, and of

these, 2,766 eyes, or 71.5 per cent, belonged to the patients with mild myopia, and 1,102 eyes, or 28.5 per cent, to those with rapidly progressive malignant myopia. Among the patients who did not accept a stronger correction than a 5.75 diopter lens, 1,847 eyes, or 66.7 per cent, were normal; 725 eyes, or 26.2 per cent, had a vision of 50 per cent of normal and 194, or 7.1 per cent, had a vision of less than 50 per cent. A study of the patients with advanced myopia showed a decided reversion in the numbers and the acuity of vision, 1.7 per cent retaining normal vision and 75.6 per cent having vision of decidedly less than half the normal.

TABLE 3.—*Complications in Cases of Malignant Myopia*

Complications	Men	Women
High myopia (over 10 D.)	170	292
Conus myopicus	227	303
Pigmentary changes in the fundus	8	10
Choroidal degeneration	62	102
Pseudoneuritis	1	2
Chronic degeneration of the vitreous	12	15
Retinal detachment	1	3
Subluxation of the lens	1	0
Lenticular opacities	20	41
Various complications resulting in:		
Amaurosis of 1 eye	27	27
Amaurosis of both eyes	0	0
Macular changes	14	49
Anisometropia	48	54
Nystagmus	10	8
Other complications	26	52

TABLE 4.—*Complications in Cases of Malignant Myopia in the Two Sexes* *

	Men	Women
Astigmatism (corneal and lenticular)	31.0	33.6
Various choroidal changes	7.0	7.9
Macular changes	1.4	3.8
High myopia (over 6 D.)	19.3	22.6

* These figures are estimated on the total number of patients with myopia.

The addition of this to the grave complications associated with malignant myopia tends to underline the difference between the two forms of myopia.

The data in table 4 show that on the whole men have been more fortunate than women, as in them the disease not only occurs less frequently but tends to run a milder course.

In order to understand the gravity of high myopia it is important to add that 72.3 per cent of persons with this form of myopia show some sort of abnormality in the fundus of the eye.

In discussing the question of what other important factors besides near work and accommodation influence the progress of myopia, let me briefly review the ophthalmologic literature on the subject.

The general opinion of ophthalmologists, as expressed by Bartels,[1] Braun, Heinonen,[2] Federici [3] and Steiger,[4] is that myopia is inclined to be associated with the economically better placed classes. Steiger based his opinion on the belief that cultured persons, by virtue of their occupation and a transition from a wandering to a settled life, where near work is highly specialized and necessary, protect and favor the development of myopia.

Brana [5] added to this that the weaker constitution of the city population is undoubtedly of importance. That the question is not entirely clarified is seen from the studies of Popow [6] and Ochapovsky,[7] who found a great percentage of myopic analphabets.

The oldest authors, von Graefe, von Arlt,[8] and Donders, believed that near work produced an increase in the intra-ocular tension leading to thinning and stretching of the posterior bulbar wall. Hess and Heine [9] disproved this by demonstrating that even the maximal contraction of the ciliary muscle does not result in increased intra-ocular tension.

More recently, Levinsohn,[10] the principal experimenter on monkeys, tried to prove his mechanical theory that myopia is caused by stretching of the eye in predisposed persons due to bending of the head and body forward and down, as is common in doing near work. Bücklers,[11]

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10. Levinsohn, G.: Die Entstehung der Kurzsichtigkeit, Berlin, S. Karger, 1912.

11. Bücklers, M.: Anatomic Relationship Between Senile and Myopic Circumpapillary Choroidal Atrophy: Seven Cases, Arch. f. Ophth. **121**:243, 1928.

Scheerer,[12] Marchesani[13] and Jablonski,[14] repeating the same experiments, disagreed with Levinsohn's conclusions. Scores of authors can be named who have favored the theory that myopia is acquired and influenced by near work. Among them are Teleky and Thier,[15] Hirsch,[16] Kantor[17] and Duke-Elder.[18] Those who have opposed this theory are Holtzman,[19] Steiger, Incze,[20] Brana, Vogt[21] and Poos,[22] who have expressed the belief that muscular activity coincident with near work is at no times sufficient to produce a primary myopic condition. Poos based his opinion on three observations: (1) that every person must accommodate in his near work, yet comparatively few are myopic; (2) that there are many families of workers with progressive myopia who have learned neither to read nor to write and who perform scarcely any near work, and yet their myopia progresses, and (3) that myopia is frequently unilateral, the normal eye remaining normal, despite near work, while the myopic eye becomes amblyopic and squinting and the degree of myopia constantly increases.

One sees, therefore, that opinions of the influence of near work on myopia are still divided. As to the question of heredity, Vogt was the first to point out its importance. He argued that the majority of degenerative changes in the myopic fundus are not the result of stretching of

12. Scheerer, R., and Seitzer, A.: Myopic Changes of Ocular Fundus in Various Degrees of Refraction, Klin. Monatsbl. f. Augenh. **82**:511 (April) 1929.

13. Marchesani: Sitzungsbericht d. bayerischen augenaerztlichen Vereinigung, 1929.

14. Jablonski, W.: Ueber den augenblicklichen Stand der Erforschung der Kurzsichtigkeit, Zentralbl, f. d. ges. Ophth. **28**:129 (Nov. 8) 1932; Zur individualisierenden Indikationstellung bei der Brillenverordnung, München. med. Wchnschr. **72**:1500, 1925.

15. Teleky, L., and Thier, A.: Beiträge für Frage der Berufsmyopie, Arch. f. Gewerbepath. u. Gewerbehyg. **1**:240 (Feb. 14) 1930.

16. Hirsch, L.: Die Berufskrankheiten des Auges, München, J. F. Bergmann, 1910.

17. Kantor, D. W.: Zur Myopiefrage der Schriftsetzer in nationalem Aspekte, Ztschr. f. Augenh. **72**:384 (Nov.) 1930.

18. Duke-Elder, W. S.: An Investigation into the Effects upon the Eyes of Occupations Involving Close Work, Brit. J. Ophth. **14**:609 (Dec.) 1930.

19. Holtzman: Deutsche optische Wchnschr., 1926.

20. Incze, A.: Ueber die Myopie als eine konstitutionelle Veränderung, Ztschr. f. Augenh. **67**:20 (Jan.) 1929: Körperbau und Refraktion, ibid. **66**:50 (Sept.) 1928.

21. Vogt, A.: Parallel Between Senile and Myopic Degeneration of Eyeball, Schweiz. med. Wchnschr. **54**:429 (May 8) 1924; Ueber Berührungspunkte der senilen und der myopischen Bulbusdegeneration, Klin. Monatsbl. f. Augenh. **72**:212, 1924.

22. Poos, F.: The Myopic Problem, Arch. Ophth. **2**:66 (July) 1929; Development of Eyeball Under Influence of Increased Field of Gravity; Experimental Study of Mechanical Factors in Genesis of Myopia, Arch. f. Ophth. **125**:1, 1930.

the eye, that these changes appear years after the growth of the eye has terminated and that, furthermore, changes similar to those hitherto considered as specific for myopia occur in hyperopic eyes.

Concerning the most important problem, that of the influence of full correction on the progress of myopia, one sees that the opinions are again divided. Whereas the older school, with Heine [23] as its leader, asserted that full correction is capable of controlling myopia, others, including Goldschmidt, Waetzold [24] and Schall,[25] have stated that full correction is useless in checking the progress of myopia but, on the contrary, shows a tendency to favor the advancement of myopia. Mekler, being more cautious, expressed the belief that despite the fact that full correction does not influence the progress of myopia, it still should be prescribed and wearing of the correction insisted on, because it at least relieves the convergence stress so common in patients without correction.

Jackson,[26] in his study of 77 myopic patients who had worn full correction for three years, found that 45 per cent showed no increase in myopia, 34 per cent showed an increase less than 1 diopter and 21 per cent an increase of more than 1 diopter.

Jablonski, in his opinion as to the value of full correction in checking the progress of myopia, is definitely against those refractionists who determine the strength of the correction in an objective manner and then order the patient to wear this correction regardless of his subjective feelings.

ANALYSIS OF MATERIAL PRESENTED

Of 2,166 myopic patients, a group of 772 who came several times to the clinic could be separated and carefully studied.

In analyzing this group it was found that some of the patients showed no advancement of myopia, some showed only a mild progressiveness and some had "progressive malignant myopia."

The incidence of the various types is presented in table 5.

Of the group of 772 carefully examined, the condition in 207 remained stationary at least during the period studied and in 565 it was progressive. In the group with progressive myopia, 220 were found to have the "malignant progressive" type. In other words, of this group of patients examined 27 per cent showed no advancement of myopia, 44.6 per cent showed a mild progression coming to a standstill before 6 diopters of myopia was reached, and 28.4 per cent showed

23. Heine: Klinisches und Theoretisches zur Myopiefrage, Arch. f. Augenh. **49**:14, 1903.

24. Waetzold: Ber. u. d. Versamml. deutsch. ophth. Gesellsch., 1931.

25. Schall: Zur Frage der Vollkorrektion bei Myopie, Klin. Monatsbl. f. Augenh. **72**:243, 1925.

26. Jackson, E.: Control of Myopia, Am. J. Ophth. **14**:719 (Aug.) 1931.

such an advancement that when the process finally came to an end so much damage had been caused that little if anything could be done for the patient.

This, of course, occurred in persons who wore the prescribed correction continuously. Not only has this correction not shown any influence on the higher grades of myopia, but even the milder form seemed to go its way, perhaps increasing for from two to three years or more after the initial examination, to come to what seemed to be a predestined halt. It is this failure to influence myopia with proper correction that leads me to believe that the cause lies much deeper and is more complicated than the common belief that near work and accommodation are mainly responsible for the development of myopia. If it were really so, how could one explain the cases of grave and rapidly progresssing myopia seen in children, in whom pathologic

TABLE 5.—*Incidence of Types of Myopia*

	Male		Female			
	Cases	Percentage	Cases	Percentage	Total	Percentage of Total
Stationary myopia	122	15.9	85	11.1	207	27.0
Progressive myopia (not over 6 D.)	141	18.2	204	26.4	345	44.6
Progressive malignant myopia (over 6 D.)	70	9.0	150	19.4	220	28.4
Total	333		439		772	100.0

changes originate early and develop far too rapidly in proportion to their near work and accommodation? Proper correction in these cases has shown no influence on the progress of myopia.

It is my personal belief that the most logical explanation presented so far, although very little investigated up to now, is the heredobiologic theory advanced by Hess, Siegrist [27] and many Russian authors, such as Ochapowski, Cholin, Cardo-Sisoev and others. In main, their theory is (*a*) Myopia is a process the extent of which is cast in the embryonal life. On the whole, it will, as a rule, run its predestined course guided by the same stimuli and laws that govern the general body growth. (*b*) In particular, it may be in a way influenced directly by the inherited strength of the fibroblasts of the sclera and some other as yet unknown factors and indirectly, to a lesser degree, by the general state of health and the conditions of work and occupation.

CONCLUSIONS

1. The real cause of myopia is as yet unknown, the most plausible being a heredobiologic process.

27. Siegrist, A.: Refraktion und Akkommodation des menschlichen Auges, Berlin, Julius Springer, 1925.

2. Myopia usually comes to a standstill at the adolescent age, excluding progressive malignant myopia, which, once started, continues to progress. In this case the influence of correction is at least doubtful.

3. That myopia is more prevalent and more severe in women is an established fact, although, as a rule, men in the same class use their eyes more strenuously.

4. Complications are just as common in persons wearing full correction.

5. Despite proper correction, the condition showed a definite tendency to progress in 73 per cent of the patients on whom follow-up study was made.

6. That advanced progressive myopia is grave is seen from my statistics, which show that 72.3 per cent of the patients had pathologic changes in the fundus of the eye and only 1.7 per cent retained normal vision.

7. The ophthalmologic profession is at present divided into two camps: those for and those against full correction in myopia, some even claiming that full correction aids the progression of myopia.

8. I believe that the wearing of correction is of great importance in aiding the physical and cultural development of the patient, although this conclusion is irrelevant to the question at issue.

PROVISIONS FOR THE SCHOOLING OF THE BLIND AND PARTIALLY BLIND

EDWARD M. VAN CLEVE
Principal of the New York Institute for the Education of the Blind
NEW YORK

When the skilled ophthalmologist, despite his faithful use of all his powers, finds his patient losing such vision as he may have; or when the operation which had been indicated and was performed has resulted disastrously; when blindness is imminent or has actually come, the question is always "What next?" The patient himself may ask it, and the great-hearted physician wishes he might give comfort and encouragement in the face of disaster. If the patient is an adult and a professional man, let him know that if sight is lost all is not necessarily lost, for there are many ways in which blind men and women may live happy and useful lives. To help in just such crises there have been formed such associations as the New York Association for the Blind, commonly known as the Lighthouse, the Industrial Home for Blind Men, and the Marie Bloede Workshop for Blind Women of Brooklyn, the Queensboro Workers for the Blind, and others, in this great city.

But if the question "What next?" is asked by the parents of a child there is at once a very prompt and properly acceptable answer, and it is particularly to the formulation of this reply that I am to attempt on this occasion as comprehensive treatment as time and your patience may afford. Schools for the child who is blind or has vision too greatly reduced to make its use in reading books of ordinary-sized type practicable are provided, and an education as good as the seeing brother and sister may obtain, or even a better education, for that matter, is afforded through the intervention of the state or city or private enterprise. The ophthalmologist should know what means there are for making the child's chance for schooling immediately available and where he may advise the parents to turn for securing the special training called for by loss of vision. In our city there are: the New York Institute for the Education of the Blind, the organization longest in the field, the first school of its type to open in the United States, founded in 1831, beginning its work in 1832, and continuously serving its purpose for nearly one hundred and three years; the public school classes for the blind; the Catholic Institute for the Blind; the Jewish Guild's Yonkers Home. Too often a child who has lost his sight is, through the ignorance of parents, denied

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Read before the Section of Ophthalmology of the New York Academy of Medicine, Feb. 8, 1935.

the privilege of having a schooling that will give him a chance to win a position of happiness and usefulness in the community. Yet there are the means ready at hand.

Again, through the enlightened services of organizations such as I have named and under leadership provided through the educational activities of the National Society for the Prevention of Blindness, there are arrangements of a special nature for the schooling of children of low vision in the so-called sight-conservation classes of the public schools. There is a stage of visual acuity and ocular condition at which the ophthalmologist advises less use of the eyes than the ordinary school-room work requires; perhaps 20/100 is the best vision the patient has and he is able to read Jaeger's test type 6—he is not blind, yet he should use his eyes with extreme care. For him and others with various degrees of vision and various conditions of the eyes the city provides classes where in small groups and with special teachers a kind of training may be had which will serve his neeed and save his visual powers from further deterioration. A special technic in this field has been developed in this city and devoted friends of the movement, along with city physicians, make possible the routing of children who should attend sight-saving classes instead of continuing to struggle to get on in the regular classes of seeing children. This subject of conservation of vision calls for more detailed consideration than can be given here.

I turn, then, to the blind child, who must use for his schooling some other means of knowing the world and its ways than those possible through vision. The parents have been brought to believe that all has been done for him by the professional man that can be done, and the ophthalmologist has advised the special school and given the parents the name and address of the principal. An interview is arranged with the chief teacher, and this reveals to the parents that Harry has before him, even though he is visually underprivileged, the opportunity to develop his whole nature—head, hands, heart, body—and arrangements are made for his entrance on a course of training that may last ten, twelve, fourteen years and result in a well rounded, efficient, satisfactory preparation for meeting manhood's problems.

WORK OF THE NEW YORK INSTITUTE FOR THE EDUCATION OF THE BLIND

Who may be admitted to the New York Institute for the Education of the Blind? We have one general rule: We accept any child whom we believe ourselves able to help, with no distinction of race, creed or social condition. He must be blind or have such defective vision as to make schooling in the usual way too difficult for him; he must be mentally capable, in reasonably good health and have, too, good moral character; and, a very important qualification, he must be *willing* to come to us and to do his best. One reason why we maintain so high a rating in

school accomplishment is because no child is ever *sent* to us, that is, compelled to attend by court or truant officer; he comes because his parents and he wish to make use of the rather unusual facilities we have to serve him. We provide schooling and board free; usually our pupils live so near that they go home for the week-ends, thus making possible for them intimate and continuous touch with the home, their friends out of school, their church. We are very chary of our character as a school—strictly and solely a school; we are not an institution in the narrow sense of that term, only in its large and generic sense. We assume custodial care of no child. Our function is no different from that of the first-class boarding school in which parents enter their children for training. There is no "institutionizing" of our young people. It is a free school, however, and no charge is made for tuition or board. It is supported by voluntary contributions, by income from gifts and legacies accumulated through nearly one hundred years, and by per capita allowances from the states of New York and New Jersey at present and a very small annual per capita contribution of the city of New York made to us as a corporate school.

Before a pupil is received we are assured that he is eligible as a blind or partially blind person through the very generous assistance of our ophthalmologist, Dr. Bernard Samuels, who sees all candidates and either makes examination de novo or confirms the reported visual condition given to the principal by the parents who make application. Let me also say that as principal of the institute I am indebted to the members of the Section of Ophthalmology of the New York Academy of Medicine for helpful cooperation in connection with the admission of children who have been their patients.

It is through the examination of our pupils by our ophthalmologist that I am able year by year to make up reliable statistics as to causes of blindness. In many instances schools like ours lack the expert analyses which we fortunately have and, therefore, their statistics are not so accurate or worth while.

It is our privilege to take the blind child of 7 and through such training as has been found most appropriate for him to fit him for life's contacts and achievements. It would be advantageous were some preschool training of an intelligent character provided; parents of a blind child are not always wise and usually in tenderness spoil the so-called afflicted one; we welcome, therefore the opportunity to advise with the parents of a child who will be obliged to use the facilities of our school if we can become acquainted with the child and them a year or more before the age of entrance. Likewise, when the incidence of blindness or partial loss of sight occurs in later childhood, it is an advantage for us to know the situation before we receive the pupil. The ophthalmologist will serve his patient and us if he will but bring us together ahead of the absolutely needful time.

What does a school for the blind do for its clientele? In general, it follows the courses of study for sighted pupils of the public schools, and the institute carries on its service through the usual elementary grades and through the high school to prepare such as are capable even for college entrance. Our pupils take the examinations of the regents of the University of the State of New York, the only allowance made for their sightless condition being the provision of an amanuensis when necessary, and the record of achievement in this particular of scholarship is one of which the institute is properly proud. And, I may add, our pupils who have gone to college are for the most part successful in their pursuit of higher education. But the school does more than train brains; it recognizes the obligation to develop body, hand and spirit.

Physically a sightless child is likely to be subnormal and needs exercise and directed development even more than the seeing one. From the first and throughout all the years of his schooling the blind boy is under the regimen of well organized gymnasium training, playground exercise and athletic sports, so that physically he becomes quite as capable as if he saw. Our young fellows hold field meets and wrestling matches with teams of seeing boys and win or lose like the good sportsmen they are.

Manual training in the institute is much more extensive in program and achievement than in the regular school; we give more time to it; we emphasize and appreciate its special value for blind people. Many of the things we seeing people do are pure imitation, and we do not need to be taught; the sightless student lacks this help which is inspiration and spur as well as pattern; he must be taught to handle his world of things. This is quite evidently necessary in his mastery of tools and their use. People see our boys fashioning articles of wood or iron or what not and exclaim in wonder; the admiration is rather misplaced—not what is made but the pupil's power and willingness to receive the training and the ability, patience and skill of the teacher are the appropriate objects of amazement. Weaving of beautiful and useful fabrics with development of taste in their making, basketry and other forms of hand work are taught; then our girls become proficient with the needle and the sewing machine and with the other tools of women's hand activities, and they learn to cook, too; and by all these means they make themselves efficient members of the family when they are at home.

In the field of education in art, the region in which the blind may be best exercised is that of music. Obviously any artistic development or expression that demands use of vision is out of their sphere. The great majority of people enjoy some form of musical outpouring, and the blind are not different from others in this particular. Perhaps because of their special limitation they are more susceptible to this means of emotional approach. But blindness does not render a girl or a boy more capable of producing a good quality of musical expression. Not all blind

children are capable of becoming musicians. As music is, however, a chief channel of artistic expression for the sightless, the institute provides that all the children may have opportunity to develop any latent powers of either appreciation or performance, and for the really musical a course of instruction through eight or ten years is available which in character as well as in extent is equivalent to a musical conservatory's program. Our public performances of musical compositions of even the highest class, such as the works of Beethoven, Chopin and Brahms and, most lately, the Christmas section of Handel's "Messiah," attest the capability, the ambition and the enjoyment of the best which our young people possess.

It would be an incomplete statement of what a school for the blind and the institute in particular does for its patrons if there were omitted reference to the development of the inner life. Throughout the years of schooling the pupils are subjected to influences, carefully fostered, that have for their purpose the building up of a kind of character that will appreciate and exemplify the admirable traits by which are distinguished the better and the best, the refined and cultured. Teachers who know how to deal with young people, who know more than what is in their books and who care for their pupils' growth into fine manhood and womanhood are sought out and kept in service without any pull, political or other. Housemothers who give attention to the physical needs of the children but also try to train them to right habits of living and thinking come into the closest of relationships with their charges and often prove most potent in developing the social qualities that all must cultivate. Religious instruction is not contemplated in the organization of a school that serves all groups of people without discrimination; but a high moral quality of spiritual intercourse is exemplified in morning and evening assembly with recognition of the Deity and our relationships to Him and to the higher things in song, reading and prayer, and in all the dealings of teachers and staff with the pupils.

What are the end-results of these efforts? Some children respond admirably and, living in school the normal sort of life contemplated in all our programs, come to be capable, happy, productive members of society. In their scholastic work they have been treated with the same consideration (and no more) as their sighted competitors in schools are shown. They take the examinations of the regents of the University of the State of New York, and through the forty-four years for which the statistics are available our students have maintained an enviable record of high scholarship. They have learned to use the typewriter and the dictaphone, to read and write with intelligence, as well as any high school boys and girls, and some have gone beyond this educational stage and entered college. Here they have usually acquitted themselves well, and a few have attained distinction, being graduated with honors,

and winning election to Phi Beta Kappa. It is not the function of the elementary and high school to produce artisans or artists, but to start the pupils on their way. Some use the hand processes they have been taught in school to do one sort or another of work when out of school; others find places where they may use their mental skill in service or personal growth and enjoyment. Specifically, in the last twenty years pupils have found occupation in these ways among others: as piano tuner, newspaper dealer, short story writer, operator of a small store, dictaphone operator and typist, switchboard operator, church organist, teacher of organ and of piano, private tutor, partner in a kindergarten, masseur, osteopath, minister of the gospel, attorney at law, director of an extensive trucking business, coal dealer, life insurance agent, radio entertainer, and social worker, especially in such service as is carried on by the Lighthouse and other like organizations.

In what I have said emphasis has been laid on the attention given to personality in our treatment of these young people; on the services that are rendered by the ophthalmologist, the teachers and the officers of the school, the state and the friends who have provided the means; on the growth and development of the children in mind, body, talent and spirit. Perhaps a few words should be said about equipment. Visitors to the institute are always very favorably impressed by the appearance of the buildings, by the beauty of their situation and surroundings, the campus and its landscaping. Looking further, they may see how admirably the needs of the blind children have been anticipated in the equipment with tools and appliances: the playgrounds have apparatus for free enjoyment and for directed physical training and athletics; the wood-working and iron-working rooms and the other manual training departments are places of well planned industry; there is a great library of books in braille, devices for writing both by hand and by machine, tangible maps and globes, typewriters, dictaphones, scientific apparatus, pianos (more than thirty of them Steinways, by the way, and three of them concert grand pianos), a fine pipe organ, various other devices for musical education, and a department for training young men in the tuning and repair of pianos. In short, there have been provided all the means known for complete and thorough development of the sightless children who come within the institute's walls.

ORIGIN OF THE INSTITUTE

It is a fine piece of philanthropy which was begun one hundred and four years ago by a young doctor who had already given his attention to helping the deaf, Samuel Akerly, associate and relative of the eminent physician Samuel Latham Mitchill, himself distinguished as physician and author, and Samuel Wood, Quaker, bookseller and publisher, progenitor of the Woods whose publications are recognized standard books in the medical world. Akerly and Wood secured the incorporation

of an institution to provide educational opportunities for blind children, and within a year there came into association with them a young physician who had just returned from Athens where he had given his services to the Greeks in their struggle for independence from the Turk, Dr. John Dennison Russ; this youthful, enthusiastic philanthropist offered his services as teacher and on March 15, 1832 began the first organized experiment on American ground of instructing the blind. In those days doubt was general that the blind could learn!

OUTSTANDING FIGURES IN THE DEVELOPMENT OF THE INSTITUTE

A century of service and of progress is exemplified in the record and the standing of the New York Institute for the Education of the Blind. A noble company of servants of humanity are the men and women who have been drawn into the work of making plainer the way of the blind youth; besides Akerly and Wood and Russ, there were here in New York, John R. Stuyvesant, the Ketchums—Morris and Hiram, the Woods—Dr. Isaac and John and Edward, Anson G. Phelps and the Schermerhorns—Peter Augustus, Alfred and others of this notable family of philanthropists, James Boorman, merchant prince and college founder—these as founders and supporters; then as teacher and superintendent, James F. Chamberlain, whose benign reign was the most notable after the beginnings wrought by Russ, and for more than half a century William Bell Wait, a great educator and leader of national influence. Others who helped make the century's history notable were Dr. John D. Fisher and Samuel Gridley Howe and Michael Anagnos and Edward E. Allen in Boston; Friedlander and Chapin in Philadelphia; Morrison in Baltimore; Smead in Ohio; Bacon in Illinois, and all over the land faithful and devoted personalities whose services make today's achievements possible.

We have a better chance today to make plain the path of the blind than had our predecessors. We have not only the history and experience of these hundred years; we have the cooperation of many agencies, the sincere interest of friends and upholders, a high standing in the educational world and recognition from other professions, such as this you have here given by inviting a representative of the educational phase of dealing with blindness to present his case.

AN INVITATION

Dr. Samuels, my friend and staunch supporter, has frequently asked me to receive as visitors to the institute representatives of his classes of students. He thinks they have been benefited by their contact with the work we do as they look forward to their professional career. We

very courteously and cordially invite you who have given your kind attention to this account of our work to come and see it in process. I invite you all, too, to know more about this organized tool of helpfulness through reading our year-book. I know that annual reports are a nuisance too often and reading them a weariness to the flesh; but in my story of the progress of our school's work I try to tell each year a bit of what is being accomplished and the pictures usually tell an interesting story; so I am not too reserved in recommending to you a perusal of our handsome year-book. We wish you to know the institute and be willing to send us promising pupils all the while.

Clinical Notes

PRERETINAL ARTERY

HAROLD F. WHALMAN, M.D., LOS ANGELES

Goldstein and Wexler [1] prepared an anatomic study of the preretinal artery, reviewing reports of thirty-two cases and describing a case under their observation in which autopsy was performed.

Early observations did not differentiate the preretinal artery from the embryonal remains of the hyaloid artery, and it remained for Hirschberg in 1885 to make the distinction. In the majority of cases

Fig. 1.—Preretinal artery of the left eye.

reported the loop has been arterial. In some cases the loop is connected with vessels on the disk, and less frequently they enter the disk independently. The condition may be bilateral, or it may occur in both the vein and the artery of the same eye.

REPORT OF A CASE

A Jewish man aged 21, had myopia of 5.5 diopters; vision was corrected to 20/20 in the right eye and to 20/15 in the left eye. Near-sightedness was the only complaint. The patient had not noticed any scotomas.

From the Department of Ophthalmology, University of Southern California Medical School.

1. Goldstein, I., and Wexler, D.: The Preretinal Artery: An Anatomic Study, Arch. Ophth. **1**:324 (March) 1929.

In the right eye the inferior temporal branch of the retinal artery emerged from the lower quadrant of the disk into the vitreous humor for a distance of about 1 disk diameter and twisted on itself in four spirals, returning to enter the retina at the lower margin of the disk, where its distribution was like that of the inferior temporal branch of the retinal artery.

The left eye presented a similar picture, except that the spiral was about half the length of that in the right eye, the inferior temporal artery was quite small,

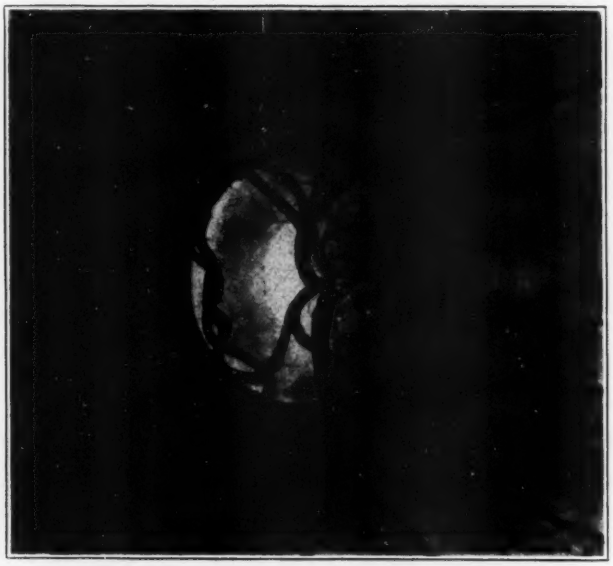

Fig. 2.—Preretinal artery of the right eye.

and there was an additional branch from the superior temporal artery of normal size distributed to the inferior temporal region.

727 West Seventh Street.

NITROCELLULOSE OF LOW VISCOSITY USED AS AN EMBEDDING MEDIUM FOR EYES THAT ARE TO BE SECTIONED

W. E. Fry, M.D., Philadelphia

Ruby in 1933 called attention to the fact that through developments in the lacquer industry there were available on the market products of nitrocellulose from which masses for embedding could be prepared that had certain advantages over the pyroxylin hitherto used for histologic purposes. According to Wilson, the viscosity of commercial brands of pyroxylin ranges from two hundred seconds to one-half second, as measured by the falling ball test. Ruby used nitrocellulose with a viscosity of one-half second manufactured by the Hercules Powder Company. Tissue that had remained in the nitrocellulose blocks for a year

From the Department of Ophthalmology of the University of Pennsylvania.

in his laboratory had shown no change in staining characteristics that could have been produced by the nitrocellulose.

In 1934 Davenport and Swank [1] reported on the use of nitrocellulose of the same degree of low viscosity. They found that it was possible to obtain a firmer block with less shrinkage with this medium than with pyroxylin, that the low viscosity permitted more rapid infiltration, and that the blocks could be hardened more rapidly. They also found that the mixture of alcohol, ether and nitrocellulose would tolerate as much as 6 per cent of water. This permitted the use of 95 per cent instead of absolute alcohol in preparing the embedding solution.

Recently, I have used nitrocellulose of low viscosity in preparing eyes for histologic examination. The eyes were dehydrated in the usual manner. They were then infiltrated for from twenty-four to forty-eight hours each in solutions of nitrocellulose of approximately 3, 6, 12 and 24 per cent concentration. The latter solution was used for embedding. The nitrocellulose was dissolved in equal parts of absolute alcohol and ether. The block was hardened overnight in a closed vessel; it was then exposed to chloroform vapors and finally hardened in 70 per cent alcohol.

By this method it has been possible to obtain sections of the eye regularly at 8 microns and sections of the posterior half of the eye at 4 microns. The sections were cut with a sliding microtome, the knife being flooded with 70 per cent alcohol. They are smooth, and may be handled as easily, and in the same manner, as pyroxylin sections.

As a preliminary report concerning the use of nitrocellulose of low viscosity for embedding eyes, the following advantages over the use of pyroxylin are listed:

1. More rapid and complete infiltration and embedding
2. Less shrinkage during hardening
3. Thinner sections obtainable
4. Less expensive material, the cost of the nitrocellulose per pound being approximately that of pyroxylin per ounce.

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1. Davenport, H. A., and Swank, R. L.: Stain Technology **9**:137, 1934.

Ophthalmologic Review

HARDENED OPHTHALMIC LENSES

HERMAN P. DAVIDSON, M.D.
CHICAGO

Accident prevention in the United States has been pushed by safety engineers for years. In some past analyses [1] of industrial accidents it was found that as high as 25 per cent were to the eyes. It was easy to provide side-screens, but ordinary ophthalmic lenses gave only limited protection. Large and small particles, coming with force, shattered these lenses, and often the cutting edges of the shattered glass added to the danger by inflicting additional wounds.

UNSUCCESSFUL RESEARCH

Substitution for the lenses was attempted: Fine wire mesh, isinglass, two lenses cemented together, isinglass and one ophthalmic lens together and wire mesh with one ophthalmic lens were all tried and discarded as unsuitable for this use. The formula of ophthalmic lenses could not be altered to accomplish any hardening process in a transparent medium. The workers therefore returned to the original formula.[2] Ordinary ophthalmic glass is a "soda lime" crown glass, purified by removal of some of the iron oxide.[3] The problem was to harden this glass without removing the transparency.

THE HARDENING PROCESS

There is a secret process which each glass manufacturer guards zealously. However, each one has apparently the same process. Blanks of the base curve desired are placed in a heavy oil and brought up to a certain degree of heat. This rearranges the molecules so that they become resistant to blows and if the glass is broken the pieces remain

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Review made at the request of the Committee on Standards, Section on Ophthalmology, American Medical Association.

1. Davidson, H. P.: Good Eyesight in Industry, Publ. 87, New York, National Society for the Prevention of Blindness, Inc.

2. O'Brig, T. E.: Modern Ophthalmic Lenses and Optical Glass, Philadelphia, Chilton Company, 1935. (This article gives an excellent review with illustrations and is recommended for reading.)

3. Coblentz, W. W., and Stair, R.: Transmissive Properties of Eye Protective Glasses, Technologic Paper 369, U. S. Dept. Commerce, Bureau of Standards, 1928.

together and do not fly. This process is described as "heat treating" or "case hardening." The glass is erroneously called "nonshatterable" glass. Each glass manufacturer gives the processed lens a name suggesting hardness; one calls its product "super armor plate" lens; another designates it as "hercules" lens, and so on.

Just how this heat treatment causes the hardening is not known, but a homely illustration may convey the idea: Place each of your hands in front of you with the fingers extended, spread apart and pointing toward each other, but not interlocking, and have some one strike your hands. The hands will have considerable movement and unsteadiness. Now interlock the fingers and have the blow on your hands repeated. There is much less motion and unsteadiness, because one hand aids the other.

PRECAUTIONS FOR GRINDING PRESCRIBED LENSES

The "heat-treated" lens is hardened for varying depths in the lens, but the center is still unhardened ophthalmic glass. For this reason, any minus lens stronger than 1.75 diopters or any plus lens stronger than 4.5 diopters should not be ground from an ordinary blank, as the cut surface in grinding may extend down to the area of soft lens. For corrections stronger than these, especially molded blanks of the proper shape for the particular correction are used. In this way no soft area is exposed. These lenses are polished and put out with the same care as are all other prescriptive ophthalmic lenses.[4] They are between 2.8 and 3.6 mm. thick, to satisfy government specification, and for prescriptive work the lens 45.8 mm. in diameter is best. The lens of smaller diameter, with side-screen goggles, does not give enough side vision, and the 50 mm. lens is rather heavy for prescriptive work when the government requirement for thickness is adhered to. The pupillary distance must be accurately measured and the proper decentration made in all lenses. A cement bifocal segment can be put on the inside for presbyopes. One piece bifocals can also be made, but the cost of supplying them is much higher.

GOVERNMENT SPECIFICATIONS

Government specifications for plano lenses are as follows:

GGG-G-501: Dec. 9, 1930:

E-6a. *Eyecup goggles* shall be fitted with two clear glass lenses as described below. Lenses shall measure 1.968 ± 0.004 inch (50 ± 0.1 mm.) in diameter, and not less than 0.11 inch (2.8 mm.) nor more than 0.14 inch (3.6 mm.) in thickness.

4. This information was obtained in a conversation with a representative of the American Optical Company, Mr. Parkenheimer.

E-6b. Lenses shall be made from single solid glass plate of a grade suitable for optical use, and which will transmit not less than 89 per cent of the incident visible light. Glass shall be of a high shock-resistant quality.

E-6c. Lenses shall be free from stria, waves, or other visible defects which will impair their optical qualities. The optical surfaces of the lenses shall be well polished and free from visible surface defects. The optical surfaces shall not be effectively out of parallel by more than 4 minutes of arc (0.058 prism diopter). Lenses shall not be negative in refractive power in any meridian, shall not have a greater positive refractive power in any meridian than 0.13 diopter, and shall not have a greater difference in refractive power between any two meridians than 0.05 diopter. The edges shall be beveled to a crown and shall be dull finished. All lenses shall be suitable for continuous use by the wearer without discomfort or optical impairment to the normal eye.

E-7. All goggles, eyecups, and lenses shall bear some permanent distinctive marking by which the manufacturer may be readily identified.

F-2e. (1) Six lenses out of each lot of 50 or fraction thereof shall be subjected to the drop tests below. If one out of the six lenses tested fails during the F-2e (4) or the F-2e (5) test, four additional lenses of the same lot shall be tested and if one of these lenses fails, the entire lot represented by that sample shall be rejected. If the lens tested under F-2e (6) fails, the entire lot represented by that sample shall be rejected.

F-2e (4). The eyecup with lenses shall be supported on a wooden block of such size as to fit into the eyecup securely but not touching the lenses. A 5/8 inch (15.9 mm.) steel ball, approximately 0.565 ounce (16 Gm.) shall be freely dropped from a height of 39 inches (1 m.) onto the horizontal outer surface of the lens. The edge of the lens shall not chip from the shock of the impact.

F-2e (5). The lens shall be removed from the eyecup and placed flat on the end of a wooden tube having an internal diameter of 1.77 inches (45 mm.) and a rim to fit the lens. A washer of rubber packing, not more than 1/8 inch thick, and of the same internal diameter as the tube, shall be placed between the lens and the tube. The 5/8 inch steel ball mentioned above shall be freely dropped from a height of 39 inches (1 m.) onto the horizontal outer surface of the lens. The lens shall not fracture from the impact of the steel ball.

F-2e (6). One of the six lenses tested under F-2e (4) or F-2e (5) shall be broken by progressively increasing the height of the drop of the 5/8 inch steel ball. This lens shall break normally with radial cracks. Any tendency to break with concentric lines of cleavage or cleavage parallel to the surface indicates an unsatisfactory hardening process.

While this is the government standard of strength, it is universally understood and accepted by glass manufacturers that these lenses should withstand the blows of a 5/8 inch (1.59 cm.) steel ball dropped from a height of 72 inches (1.8 meter). If they do not withstand the test at 72 inches, even though the government requirement is only 1 meter, they are rejected as unsafe.

The outer surface of the lens is usually stronger than the inner, and the government takes cognizance of this by specifying that the outside shall be struck by the steel ball. If the lens is "heat-treated" properly, why are not both sides equally strong? The oil is heated to the same temperature on both sides. This difference may be explained by means

of a comparison with the baseball bat. When one is batting a pitched ball the grain of the wood must be held correctly to keep the bat from breaking.

STANDARD SIZE AND BASE CURVE

The plano lenses are round and can be obtained in any base curve desired. The 1.25 and the 6 diopter base curve are the ones most frequently used. The 6 diopter base curve is the better. The standard diameters are 45.8 and 50 mm.

HARDENED TINTED LENSES

Welding has increased immensely in the trades, and in order to protect workers from stray rays in the vicinity, a hardened lens called "calobar" is now on the market. This has all the properties of any hardened lens and is green. The medium shade has a density of 0.28, transmits 53 per cent of visible light, and absorbs 100 per cent of ultraviolet and 80 per cent of infra-red rays.[5] This protects men observing heated boilers, rivet heaters, ladlemen and metal pourers. It is not intended for use by persons engaged in the operations of welding at higher temperatures. This lens gives a borderline of absorption and abrasive resistance and still transmits 53 per cent of visible light. Ordinary lenses transmit 91 per cent of visible light.[6]

The "calobar" lens is used extensively by the aviation corps of the United States Army in goggles with a cylindrically curved wide lens, to relieve glare and to protect the eyes and sinuses from wind.

AIR CORPS SPECIFICATION NO. 3047, MAR. 14, 1933

c. The anti-glare glass shall be that known a calobar, or its equivalent. At a thickness of 2.4 mm. it shall have a total visible transmission of 53 per cent plus or minus five per cent. There shall be no absorption bands in the visible spectrum. The glass shall have high absorption throughout the ultraviolet and near infrared regions of the spectrum, in order to protect the eye from harmful effects of intense sunlight. The dominant hue desired for this glass is sage green. Anti-glare glass of a different hue will not be accepted unless a sample pair of lenses, submitted with or prior to the bid, has been approved.

3. *Surfaces.*—The surface shall be polished in accordance with high-grade ophthalmic practice.

5. *Class I—Non-corrective Lenses:*

a. Class I lenses shall conform to Figure 1 and shall fit profile gages (left and right) which will be loaned to the contractor by the Procuring Agency. The lenses shall fit the gages snugly, without requiring forcing sufficient to cause a strain as determined in Section VI, 8, or appreciable play. A round wire 0.020 in diameter shall not enter at any point between the lens periphery and the gage.

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5. American Optical Company: Eye Protection, in Catalog 33.

6. Coblentz, W. W., and Emerson, W. B.: Glasses for Protecting the Eyes from Injurious Radiations, Technologic Paper 93, U. S. Dept. Commerce, Bureau of Standards, 1917.

A metal feeler 0.015 inch thick by ⅛ inch wide shall not enter between the lens periphery and the gage for a distance of more than ½ inch at any one place, and the intervals between such flat spots on the periphery shall not be less than one inch.

b. Class I lenses shall be 2.40 mm. plus or minus 0.15 mm. in thickness at the thickest point. They shall be ground and polished to cylindrical surfaces whose elements shall be parallel to the minor axis and perpendicular to the major axis of the lens, within a tolerance of one degree. The outer cylinder shall have a curvature of nine diopters, plus or minus ⅛ diopter. The inner cylinder shall be of such a curvature as to provide the required freedom from power. The lens shall be decentered with the optical axis 14.5 mm. plus or minus three mm. from the nasal end.

TYPES OF GOGGLES

The government gives elaborate specifications for the standards of goggles for use in work. In this review [7] only the essential features will be mentioned. There are three types, made of all metal, rubberized and hard synthetic materials, respectively. All must be resistant to disinfectants and must encase the bony orbital rim as tightly as possible. All three must have ventilation.

The all metal rigid bridge goggles, with six assorted bridge sizes and metal temples, are suitable for all impact hazards. They are the most frequently used and the best prescriptive goggles. The lenses are 45.8 mm. in diameter. The ventilation is through the metal frame at the nasal side and through a mesh screen of wire on the temporal side.

The rubberized or cup goggles are used for protection against dust, acid or fumes. They are made of aluminum, are padded with rubber and conform to the contour of the face. Each eye cup is attached to the other by an adjustable bridge, and the goggles are held on by an elastic band around the head. Slotlike ventilation is achieved through the aluminum cups. The lenses are 45.8 mm. in diameter.

The third type of goggles is made of a durable, impregnated composition, a nonconductor of heat and electricity, safe and moisture-proof. Ventilation is attained at each temple by means of a wire mesh, and air holes like saw teeth are directly under the lens. There are individual right and left eye cups, which are held together by an adjustable bridge and a head-band. These goggles take 50 mm. lenses and give a wider field. They can be used over a workman's own spectacles, but for all day wear more comfort is obtained by using lenses 45.8 mm. in diameter, ground to the proper prescription. When plano lenses can be used, goggles with 50 mm. lenses are comfortably worn.

In ordering rigid bridge goggles with 45.8 mm. lenses it is necessary to give ordinary spectacle frame measurements so that the proper bridge and temple can be selected. It is more comfortable to have a

7. Federal Specifications (U. S. Govt. Printing Office) GGG-G-501 (Goggles: Eye Cup); GGG-G-521 (Goggles: Rubber); GGG-G-541 (Goggles: Welders).

fine leather pad around the edges and under the bridge. The temples can be covered by a tubing which slips over them. The cup goggles of type 2 and type 3 are adjustable at the nose by a notched chain and lock, and the size of the head band can be altered; hence no specifications other than type and diameter need be mentioned.

In ordering hardened lenses the prescription, the pupillary distance, the base curve and the color should be given. In prescriptive goggles with lenses 45.8 mm. in diameter, segments for presbyopes are 35 by 20 mm. unless otherwise specified. The lenses and segments will be ground decentered for pupillary distance. Hardened lenses and goggles are obtained from any large wholesale optical concern. All opticians can readily obtain them.

LAMINATED LENSES

When a sheet of cellulose acetate of nearly the same index of refraction as glass is interposed between two chemically treated dust-free lenses and pressure and heat are applied, there results a glass that is known by many names, among others laminated, nonshatterable, splinter-proof, triplex and reenforced glass. This glass can be made so thin that it is only 0.5 mm. thicker than ordinary opthalmic lenses of the same prescriptive strength.[2] Because of this, laminated lenses are used when blows on the glass itself constitute the greatest danger, as in the case of prescriptive lenses worn by young children. One glass acts as a shock absorber to the other, and the laminated glass withstands blows several times as intense as ordinary glass does. Besides the fact that the lenses are chemically adherent, one lens holds the other in case of accident and keeps splinters from flying. Laminated lenses can be made in color (by secret processes), and golfers, motorists and others can wear them over ordinary ophthalmic lenses and obtain considerable protection. Laminated lenses are not intended for use in industry where blows often come with great force. These lenses are now carried by most optical companies, and all opticians can readily obtain them.

In supplying hardened lenses for industry and laminated lenses for less hazardous undertakings the great glass industry has been of real service to humanity in conservation of eyesight. While no definite statistical data can be compiled, it is safe to say that two million workmen wear hardened lenses each work day. The laminated lenses are a far more recent invention, but with the advent of the use of colored glass and the additional safety which they provide without any appreciable addition in thickness and weight, new fields are opened for their use. The parent of the small child, the golfer, the aviator, the myope who must wear his glasses for sport and many other persons are sure to react favorably toward these lenses which give added protection.

30 North Michigan Avenue.

News and Notes

DEATHS

Professor Jules Gonin of Switzerland died on June 11. He was born at Lausanne in 1870 and spent his early school years at the Collège Galliard and the cantonal gymnasium, later entering the faculty of science of the old academy and the faculty of medicine at Lausanne and Bern. In 1918 he became surgeon in charge of the Asile des Aveugles, and in 1920, professor of ophthalmology in the University of Bern. Professor Gonin will be remembered primarily for his work on detachment of the retina. His first publication on this subject appeared in 1908, but it was not until 1918 that his paper "The Anatomical Causes of Detachment of the Retina" guided oculists to the point that they have reached today. His crowning achievement, his book "Detachment of the Retina," was published in 1934. Among the societies of which he was a member were the Swiss Ophthalmological Society, the French Ophthalmological Society and the Heidelberg Society of Ophthalmology.

SOCIETY NEWS

International Association for Prevention of Blindness and International Union Against Tuberculosis.—Dr. Alix Churchill, associate secretary-general of the International Association for Prevention of Blindness and executive secretary of the International Union Against Tuberculosis, recently arrived in the United States for a series of conferences regarding policies and procedures in the fight against blindness and tuberculosis.

The blind population of the world is estimated at about 5,000,000 or 6,000,000, according to Dr. Churchill. The steadily growing interest of European educators in sight-saving classes as a means of educating children with seriously defective vision is responsible for the formation of a special class in Paris. There are now 462 such sight-saving classes in 144 communities throughout the United States.

Dr. Churchill stated with regard to tuberculosis that the main lines of the campaign against this disease were worked out in the early part of the century, and that anti-tuberculosis schemes have been developed in all civilized countries since 1905. In the United States, the mortality declined from 195 per hundred thousand inhabitants in 1900 to 63 in 1932—a decrease of 67 per cent.

Mississippi Valley Medical Society.—The first annual meeting of the newly formed Mississippi Valley Medical Society will be held at Quincy, Ill., October 2, 3 and 4. Among the clinicians on the program are: Dr. Isaac A. Abt, professor of pediatrics, Northwestern University Medical School; Dr. Fred H. Albee, professor of orthopedic surgery, New York Postgraduate Medical School and Hospital; Dr. W. Wayne Babcock, professor of surgery, Temple University School of Medicine, and Dr. Hugh Cabot, professor of surgery, University of Minnesota Medical School. They will be assisted by twenty-seven

specialists from Illinois, Missouri and Iowa, who will conduct a clinical lecture course. Detailed programs may be obtained from Dr. Harold Swanberg, secretary-treasurer, 211-224 W. C. U. Building, Quincy, Ill.

UNIVERSITY NEWS

The Council of the Royal College of Surgeons of England has appointed Dr. A. F. MacCallan as a Hunterian professor for the ensuing year. Professor MacCallan will deliver a lecture on Jan. 17, 1936, at the College of Surgeons, Lincoln's Inn Fields, London, on the "Surgery and Pathology of Trachomatous Conjunctivitis."

Dr. James W. Shields of Denver has recently been appointed professor of ophthalmology and head of the department at the University of Colorado School of Medicine. Dr. Melville Black has retired from active university work.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Conjunctiva

CATARRHAL DIPHTHERITIC CONJUNCTIVITIS. J. FRANÇOIS, Brit. J. Ophth. **19:** 1 (Jan.) 1935.

The clinical histories in five cases of a catarrhal form of diphtheritic conjunctivitis are given. The disease is found principally in new-born infants, in the first days following birth. It is usually bilateral; the second eye may be attacked rather rapidly or as late as six weeks after the first eye. The secretion is mucopurulent and of a canary yellow color. The bulbar conjunctiva is slightly congested. The skin of the lids is erythematous. Preauricular adenopathy frequently exists. A mucopurulent rhinitis is common. Corneal complications may occur.

François' conclusions are: 1. Besides the pseudomembranous forms of diphtheritic conjunctivitis there is a merely catarrhal form. 2. The latter is often met with in newly born infants and should be ranked among the ophthalmias neonatorum. 3. It shows no pathognomonic sign and has the aspect of nearly all mucopurulent conjunctivitis. 4. Because of its clinical aspect, it seems to be the most benign of all types of diphtheritic conjunctivitis; however, it may be complicated with serious corneal lesions. 5. It is refractory to the ordinary treatment, but it heals completely and rapidly under the influence of antidiphtheritic serotherapy.

A bibliography is appended to the article. W. ZENTMAYER.

ENDOGENOUS GONOCOCCIC CONJUNCTIVITIS AND KERATITIS. A. VÁZQUEZ BARRIÈRE, Arch. de oftal. hispano-am. **35:** 15 (Jan.) 1935.

After stressing the points of differentiation between autogenous and endogenous gonococcic conjunctivitis, Barrière points to a third variety in which passage of gonococci from the deep to the superficial layers of the conjunctiva may convert the latter into the former variety. He then reports a case of endogenous conjunctivitis in which there were febrile and articular symptoms with abundant gonococci in a mild conjunctival secretion and in which the local application of silver nitrate with protein and vaccine therapy curtailed the attack. On the basis of a review of the literature, he distinguishes four forms of keratitis of endogenous origin: (1) phlyctenular, (2) mixed parenchymatous and vesicular, (3) pure parenchymatous and (4) progressive marginal.

C. E. FINLAY.

Comparative Ophthalmology

RÔLE OF THE EYES IN THE STIMULATING ACTION OF LIGHT ON THE TESTICULAR DEVELOPMENT OF THE DRAKE. J. BENOIT, Compt. rend. Soc. de biol. **118:** 669, 1935.

The sexual cycle of male birds is characterized by testicular hypertrophy in winter, followed by regression in summer and fall. Testicular

hypertrophy was artificially induced in drakes by exposing them to electric illumination for three weeks. Since this effect was produced when the eyes were exposed and the body covered, but not when the eyes were hooded and the body exposed, the stated action of light probably occurs through the intermediary of the eyes. Further experimentation indicates that this stimulation of the gonads is accompanied by a simultaneous stimulation of the anterior lobe of the hypophysis.

J. E. LEBENSOHN.

Cornea and Sclera

RING ABSCESS OF THE CORNEA. P. HERMANN, Arch. d'opht. **51**: 671 (Oct.) 1934.

Hermann describes a case of ring abscess of the cornea which occurred at the end of an exacerbation of trachoma and probably followed an ulceration of the cornea. He could find no exactly similar case reported in the literature, in which only thirty such cases have been recorded. The reported cases fall into two groups: those in which the abscess followed a wound and those in which it had an endogenous or metastatic origin. He reviews briefly the characteristics of this condition as shown in the reported cases and discusses the bacteriology of the disease. He points out that few cases have resulted in cure, the great majority terminating in complete loss of the eye. Only two cases were found in which the eye was saved, those of Snell and Schoute. The eye in the case reported by Hermann was preserved but with greatly diminished visual acuity.

S. B. MARLOW.

INFLUENCE OF CHANGE IN THE FUNCTION OF THE SEXUAL GLANDS ON SEPTIC WOUNDS OF THE CORNEA. S. RINALDI, Ann.di ottal. e clin. ocul. **62**: 625 (Aug.) 1934.

Rinaldi reviews reports showing that testicular and other glandular extracts have a stimulating effect on the healing of experimental wounds in general. Castration, on the other hand, does not seem to affect healing of such wounds unfavorably. He performed orchectomy on a series of rabbits, while in another group testicular and ovarian extract was injected subcutaneously. A third group was used as controls. All the animals were submitted to a linear corneal wound with a knife dipped in cultures of Staphylococcus, which were also dropped in the conjunctival sac. In undeveloped male rabbits, castration had no definite effect on the experimental infections, as compared with those of the controls. The operation had no deleterious effect on the health of these animals, which gained weight during the experiment. Animals receiving glandular extracts showed a marked resistance to the staphylococci. Usually the wounds healed promptly without signs of infection. The general condition of the animals, on the other hand, was affected unfavorably. They lost weight and several died. The effects of similar treatment in adult male rabbits were in general the same; also the effects in females in which ovariectomy or injections of ovarian extract were performed. Rinaldi believes that a general increase in metabolism was responsible for the increased resistance in the animals given glandular extracts. He

had previously reported two cases of disciform keratitis in women with glandular insufficiency, in which the use of ovarian extract seemed of definite value.

S. R. GIFFORD.

CLINICAL AND BACTERIOLOGIC STUDY OF KERATITIS DUE TO BACILLUS PYOCYANEUS. D. GIANNINI, Ann. di ottal. e clin. ocul. **62**: 869 (Oct.) 1934.

The literature on corneal ulcers due to Bacillus pyocyaneus and Bacillus fluorescens is reviewed. Giannini observed a patient aged 26 years in whom ulceration of the right cornea occurred without any preceding trauma. When the eye was seen three days after the onset of symptoms, the whole cornea except for a narrow peripheral ring was occupied by a dense infiltrate. A hypopyon filled half the anterior chamber. To the usual local treatment were added an intravenous injection of methenamine and injections of milk. On this treatment the ulcer did not progress, but tension increased, so that a paracentesis was performed and repeated after five days. After about six weeks the eye became quiet, with a dense central scar and some posterior synechiae. Several months later iridectomy was successful in restoring a visual acuity of 1/10.

Cultures of scrapings from the ulcer on plain agar gave a pure growth of B. pyocyaneus. Broth cultures shaken with chloroform and ammonia gave an intense blue color (reaction of pyocyanine). A guinea-pig into which a culture of the organism was injected intraperitoneally died within twenty-four hours. A rabbit used for corneal inoculation acquired a rapidly progressive ulcer which completely destroyed the cornea.

The question whether a differentiation is possible between strains described as B. fluorescens and B. pyocyaneus is considered, Giannini concluding that the positive reaction for pyocyanine is diagnostic of B. pyocyaneus and is not given by B. fluorescens. The case he reports was unusual in the absence of a previous injury, which was present in most of the reported cases, and in the relatively favorable outcome, since in most cases the cornea was entirely destroyed. He believes the injections of foreign protein may have influenced the outcome in his case. A bibliography is given with the article.

S. R. GIFFORD.

TREPHINING AFTER SONDERMANN IN SERPIGINOUS ULCERS OF THE CORNEA. Y. K. WARSHAWSKY, Sovet. vestnik oftal. **5**: 428, 1934.

Sondermann's trephining of the cornea in serpiginous ulcer of the cornea is based on the principle that the wound remains open for a few days with the result that the intra-ocular tension is kept low and the nutrition of the ocular tissues proceeds under favorable conditions. Warshawsky used Elliot's trephine, 1.5 mm. wide, only when the ulcers were severe and extensive and other methods of treatment of no avail. Thirty-nine mostly middle-aged patients were operated on in the course of two years at the Baku Eye Clinic. In twenty-seven cases the progress of the ulcer was stopped, and the hypopion disappeared. In three cases the trephining had to be repeated, as the wound closed too soon. In nine cases, or 23 per cent, the ulcer progressed after the operation. No

panophthalmitis or loss of an eye was observed in any of the cases in which trephining was done. Anterior synechiae formed only in four cases.

Warshawsky believes that central corneal ulcers are the most favorable for trephining, as usually no anterior synechiae are formed. Since the cornea is thinned, the trephining must be performed very carefully in order to prevent injury of the lens.

O. SITCHEVSKA.

ARGYROSIS OF THE CORNEA APPEARING UNDER THE PICTURE OF SUPERFICIAL DIFFUSE KERATITIS. M. MIKUNI, Acta soc. ophth. jap. **39:** 19 (Feb.) 1935.

A patient 52 years of age had been treated for thirty years with instillations of silver nitrate, causing a pronounced silver impregnation of the conjunctiva and a curious superficial sandlike transformation of the surface of the cornea, dark in color. The slit lamp revealed a diffuse opacity in the region of Descemet's membrane and also many fine dark gray dots of opacity in Bowman's membrane. The parenchyma of the eye was normal. The corneal endothelium was normal. In previous cases of argyrosis of the cornea the impregnation of Descemet's membrane has been reported but not that of Bowman's membrane.

A. KNAPP.

Experimental Pathology

EXPERIMENTAL PRODUCTION OF DETACHMENT OF THE RETINA. H. WEISS and J. N. EVANS, Am. J. Ophth. **17:** 627 (July) 1934.

In the course of experiments to reproduce variations in ocular tension, Weiss and Evans found that ligating the optic nerve produced a peripapillary detachment of the retina. They describe the changes that occurred in rabbits' eyes and in one human eye which was later enucleated for sarcoma of the choroid. They expect to report on the pathology of these cases later.

W. S. REESE.

CATARACT IN RATS FED ON HIGH LACTOSE RATIONS. H. S. MITCHELL and W. M. DODGE, J. Nutrition **9:** 37 (Jan.) 1935.

An adequate diet in which, however, lactose was the sole carbohydrate and constituted 70 per cent of the ration was fed to rats. In ten weeks bilateral mature cataracts developed in 68 per cent of the rodents, and definite cataractous changes were detectable in the remainder. Control animals fed on starch, maltose, dextrose or sucrose as the sole carbohydrate exhibited no lens abnormality. Except for the cataracts, the rats on the lactose ration appeared in just as good general condition as the controls. The iris, cornea, sclera, ciliary body and retina were histologically normal.

On a 50 per cent lactose ration, only 27 per cent of the rats acquired mature cataracts, and not until after they had been seventeen weeks on the diet. On a 30 per cent lactose ration, the lenses remained normal. Rats may be peculiarly susceptible to cataracts, as neither kittens nor rabbits were thus affected as a result of high lactose feeding.

J. E. LEBENSOHN.

General Diseases

FOCAL INFECTION AND OCULAR DISEASES. G. ZANETTIN, Ann. di ottal. e clin. ocul. **62:** 588 (July); 695 (Aug.) and 786 (Sept.) 1934.

Zanettin thoroughly reviews the previous literature on the subject, especially the American literature. He describes the technic of obtaining material and making inoculations in rabbits for experimental purposes and the method of Jones for staining bacteria in sections.

His own experiments concerned 25 patients with active ocular disease attributable to focal infection in whom definite foci of infection could be found. All the patients showed a negative Wassermann reaction and no signs of active tuberculosis. As dental foci only teeth with apical granuloma were included. The ocular diseases included were chronic interstitial keratitis, scleritis and episcleritis, neuritis and progressive optic atrophy.

In one case of iridocyclitis a strain of streptococci was obtained from the tonsils which caused ocular inflammation in 8 of 9 rabbits inoculated. It preserved this apparent selectivity for ocular tissues through several animal passages. Slight congestion of the bulbar conjunctiva and iris, with miosis appearing after several hours and lasting one or more days, was often the only ocular change in the animals and was considered evidence of organ selectivity. In one patient with iritis, streptococci were isolated from the tonsils, the uterine cervix and a tooth. Strains from the latter two sources produced ocular lesions. In some animals a severe plastic iritis was produced. Thirty-three rabbits used as controls were given injections of sterile Rosenow broth, stock cultures of streptococci or streptococci from tonsils of patients without ocular disease. None showed any signs of ocular inflammation. In 57 per cent of the 26 cases studied tonsillar infection was shown and in 11 of these 15 cases the organisms isolated exhibited oculotropism. Only 18 per cent of a larger group of patients without diseases suspected to be of focal origin showed tonsillar infection.

Sections of the eyes of animals showing only congestion of the iris revealed a few cocci along the walls of blood vessels, while in animals showing severe uveitis many cocci were found in the uveal tract, in the vitreous and around the optic nerves. Even in animals showing the slighter changes, streptococci could always be obtained on culture of the vitreous. Of the 101 rabbits given injections of material from patients in whom focal infection was the probable cause, 50 per cent showed ocular lesions. Of 17 animals inoculated from these first rabbits, 16 showed ocular lesions. Of the 26 patients, 16 showed streptococci which produced ocular lesions. Nine of these strains were obtained from tonsils, 3 from teeth, 2 from the cervix uteri, while in 2 cases they were obtained from 2 different foci. The largest percentage of positive results was in cases of uveitis: 11 of 16 cases, or 68 per cent. Of 5 cases of scleritis or keratitis, only 2 showed positive results; of 5 cases of optic neuritis, 3 showed positive results. These results convinced Zanettin of the truth of the theory that focal infection explains the disease studied and of the importance of a search for foci of infection in all such cases. He advises routine bacteriologic study of suspected foci with demonstration of oculotropism for the germs isolated before surgical treatment of such foci is undertaken. A bibliography is added.

S. R. GIFFORD.

GENITOSCLERODERMIC SYNDROME AND CATARACT (ROTHMUND'S DISEASE). A. RUSSO, Ann. di ottal. e clin. ocul. **62:** 646 (Aug.) 1934.

The literature on the association of cataract with scleroderma, first described by Rothmund in 1868, is reviewed. Symptoms of endocrine imbalance and laryngeal disturbances are frequently present and the disease is often familial. Russo's patient, a man of 35, noted, first, an aphonia at the age of 15 and thereafter the voice remained rough and high-pitched. At 18, the cutaneous condition appeared on the feet, with ulcers and extensive callus formation. At 22, a visual disturbance was noted, which soon involved both eyes. When the patient was first seen by Russo, vision was, right eye, 1/60; left eye, 1/16. Disk-shaped opacities were present in the posterior cortex of each lens, also peripheral wedge-shaped cortical opacities and fine punctate opacities under the anterior capsule. The skin was everywhere atrophic with characteristic patches of thickening and discoloration, especially on the hands and feet. Almost no hair was present on the body, and the face was devoid of a beard. The genital organs were infantile. The blood calcium was low (8.6 mg.). Latent tetany was demonstrated by positive Chvostek and Erb signs. No other cases were present in the patient's family.

The similarity of the lens changes to those present in parathyroid tetany, rickets and myotonic dystrophy is noted, and the significance of a disturbance in calcium metabolism in these conditions as a cause of the lens changes is emphasized. Russo discusses the effect of disturbances of the genital organs on calcium metabolism, though insufficiency of the parathyroid glands may also be present in this and other cases and is, perhaps, the primary cause of Rothmund's syndrome. A bibliography is supplied.

S. R. GIFFORD.

A CASE OF SCHÜLLER-CHRISTIAN DISEASE. G. TSUCHIYA and T. KAKU, Acta soc. ophth. jap. **38:** 116 (Aug.) 1934.

This is the second case of Schüller-Christian disease which has been reported by ophthalmologists in Japan. A boy 4 years of age who, until the development of his present trouble, had been quite well from the time of birth, now presents a left exophthalmos, which has been progressive for one year. There is a large defect in the bones on the left side of the skull. The eyes otherwise are perfectly normal.

A. KNAPP.

General Pathology

ADHESIVE CHORIORETINITIS FROM SUPERFICIAL ELECTROCOAGULATION AND CHEMICAL CAUTERIZATION OF THE SCLERA. A. BUCALOSSI, Ann. di ottal. e clin. ocul. **62:** 1039 (Dec.) 1934.

On rabbits' eyes Bucalossi employed diathermy with a 3 mm. active electrode, a current of from 50 to 100 ma. and a coagulation time of from two to five seconds. The eyes were removed for section at intervals of from three days to one month after treatment. In another series pure potassium hydrate stock or glacial acetic acid was applied to the sclera for from thirty seconds to two minutes. With all these agents it was possible, by using proper dosage, to produce marked effects on the choroid and retina without causing necrosis of the sclera. The choroid

became hyperemic and infiltrated with round cells, the deeper layers of the retina showed atrophic changes, and a layer of exudate formed between the choroid and the retina which produced firm adhesion between them. The process was complete in about a month with production of a pigmented scar. The area of adhesion produced by diathermy was limited to the area covered by the electrode, while with the chemical agents it extended over a larger area. With the acid a more intense hyperemia of the choroid was produced. One human eye with glaucoma, destined for enucleation, was treated with diathermy. (100 ma. for three seconds) and showed changes similar to those seen in the rabbits. The chemical methods described are considered to have certain advantages over the Guist method, and it is proposed to try them clinically.

S. R. GIFFORD.

Glaucoma

PRIMARY GLAUCOMA: THE RESPECTIVE VALUES OF THE DIFFERENT FORMS OF TREATMENT OF THIS DISEASE. G. H. BURNHAM, Brit. J. Ophth. **18**: 687 (Dec.) 1934.

Burnham divides primary glaucoma into two kinds: that which is associated with noncupping of the optic disk and that which is found with cupping of the optic disk. The noncupping variety passes through many stages terminating in the acute inflammatory type or, as a second type, painless gradual loss of vision. Both types of this division show every degree of plus tension from the least to the greatest. Since in the long-standing high plus tension of acute sympathetic ophthalmia there is no cupping of the optic disks, the conclusion might be drawn that plus tension cannot be held responsible for the cupping of the optic disk unless one ignores much palpable evidence to the contrary. The variety of glaucoma associated with cupping of the optic disk "is not due to cyclitis to begin with but rather its origin is owing to an inflammation of the optic disc and of the nerve immediately behind it. Later on it gradually involves the ciliary region, more in some cases than it does in others."

The treatment by physostigmine and operation is unsatisfactory, so that the author has given up this mode of treatment, except iridectomy and internal treatment in the very last stage of acute glaucoma. Cases of the noncupping type of glaucoma, with few exceptions, can be cured in the beginning by the use of physostigmine, mercury and sodium iodide and sodium bromide. If, however, the improvement is not satisfactory, the use of "the combined treatment" is advised. This consists in the administration of pilocarpine hydrochloride hypodermically for twenty consecutive days followed after an interval of six weeks by daily injections for ten days, and this course is pursued until the treatment is ended. During the intervals other drugs are given internally, including mercury and chalk, ipecac and opium, and sodium iodide. For details of this treatment a previous issue of the ARCHIVES OF OPHTHALMOLOGY may be seen (Burnham, G. Herbert: Combined Treatment in Certain Diseases of the Eye, **11**: 237 [Feb.] 1934).

(This article has been abstracted for the same reason that it was published in the *British Journal of Ophthalmology* as stated in the December issue of that journal under the heading "Annotation," p. 704.)

W. ZENTMAYER.

Is Primary Glaucoma an Inflammatory Condition? E. Redslob, Ann. d'ocul. **172:** 1 (Jan.) 1935.

Redslob states that during the course of chronic glaucoma, called primary, he has often observed on the posterior surface of the cornea the formation of precipitates. They are very fine and are seen only with the slit lamp. These precipitates are not, as has been suggested by different writers, the sign of an intra-ocular inflammatory process. They are due to a disturbance of the circulation, especially to a venous stasis which has developed in the center of the iris. It is possible, too, that in nonglaucomatous disorders the presence of precipitates does not indicate the presence of an inflammation but rather a condition of stasis.

Redslob states, also, that the blood in retinal hemorrhages may have its source in the vessels of the choroid and not exclusively in the vessels of the retina. Finally he has seen that the canal of Schlemm may contain blood in cases of primary glaucoma, intra-ocular tumors and very acute iridocyclitis.

Seven observations are reported, and the report is illustrated with photomicrographs.

S. H. McKee.

Hydrocephalus with Glaucoma in an Infant. P. Pesme, Arch. d'opht. **51:** 524 (Aug.) 1934.

Pesme suggests that this unusual case of hydrocephalus and glaucoma in an infant—ocular and cerebral glaucoma—should draw attention to the relationship which may exist between disturbances of the intracranial tension and the tension of the eye. The case of a child who died at the age of 11 months is fully described from both the clinical and the anatomic standpoint. In view of the known relationship between the retinal circulation and the intracranial pressure he propounds the hypothetic question as to what happens when there is only a partial defect of the normal drainage system of the eye and the brain. He calls attention to the frequency of high myopia in hydrocephalus and Crouzon's disease. The next step is the intimation that myopia may be dependent on mild intracranial hypertension, manifested possibly by the headaches which are of frequent occurrence in progressive myopia.

S. B. Marlow.

Substitution of Physostigmine and Pilocarpine by Arecaline. T. Krylov, Sovet. vestnik oftal. **6:** 115, 1935.

Krylov gives a preliminary report on the use of arecaline in glaucoma.

Arecaline is an alkaloid obtainable from betel nuts and is used as a laxative in veterinary practice. A 1 per cent solution of arecaline bromide causes a mild congestion of the bulbar conjunctiva and a contraction of the pupil which lasts from two to three hours.

Tables in which the comparative action of physostigmine, pilocarpine and arecaline are given show that arecaline is about ten times weaker than physostigmine and five times stronger than pilocarpine. The use of arecaline causes a transitory disturbance of color sense, as the objects appear grayish. The spasm of accommodation is about 8 diopters. The

intra-ocular tension was lowered considerably in all patients, in some to a marked degree; no complications were observed for a period of two years.

Since there is a lack of physostigmine and pilocarpine in the Soviet Socialist Republics, Krylov believes that these miotics could be safely replaced by arecaline.

O. SITCHEVSKA.

Injuries

CROCHET HOOK THROUGH BOTH ORBITS. W. L. BENEDICT, J. A. M. A. **104:** 305 (Feb. 2) 1935.

The patient was a married woman, aged 55, subject to a maniacal depression psychosis. With suicidal intent she thrust a no. 12 crochet needle from the right temporal region through both orbits. It passed through the right temple until the sharp point struck the left temporal orbital wall. Using her shoe as a hammer, she drove the hook farther into her head until the handle was entirely buried under the skin in the right temporal side. The eyes showed no abnormalities except a few retinal hemorrhages and edema of the conjunctiva. A roentgenogram disclosed that the point of the hook had been deflected sharply on the handle about half an inch from the end. This necessitated the withdrawal of the needle from the left side. A portion of the left temporal wall was removed by a rongeur. The needle was then grasped with a strong forceps and pulled on through in the direction in which it had been started.

W. ZENTMAYER.

EMERGENCY TREATMENT OF RUPTURE WOUNDS OF THE GLOBE. DUVERGER, Arch. d'opht. **52:** 5 (Jan.) 1935.

Injuries due to violent pressure on the globe are almost always followed by rupture of the sclera. They almost always occur about the limbus, although in exceptional instances they may involve the posterior segment of the eye not far from the disk. In this discussion Duverger limits himself to the former type. These are divided into injuries in which the conjunctiva is torn and the tissues thus exposed to infection and injuries in which the conjunctiva remains intact. The consensus of most authorities is that when the conjunctiva is ruptured immediate repair or enucleation is indicated, and that when the conjunctiva is intact it is in order to let well enough alone. After careful observation Duverger dissents from this view and believes that in all cases the eyeball should receive early surgical treatment whether the conjunctiva is intact or not. He believes that the fear of infection is unjustified, especially when the infrequency of its occurrence following operations for cataract and other surgical procedures in which the eye is opened is considered. Furthermore the conjunctiva has been bruised and may contain fine fissures through which infection may occur. He believes that ruptures heal more quickly and with fewer late complications if proper early operation is done. He outlines his method of procedure. The most difficult part of it is the placing of scleral sutures.

S. B. MARLOW.

Lens

CATAPHORESIS IN THE NORMAL AND IN THE PATHOLOGIC LENS. S. KREIMER and J NORDMANN, Compt. rend. Soc. de biol. **118:** 83, 1935.

In determining the iso-electric point of tissues by cataphoresis, the technic of Vlés and de Coulon is the method of choice. In the normal lenses of cattle and rabbits the iso-electric point thus determined is most generally within p_H 5.2-6.8. In the naphthalene cataract of rabbits and the senile cataract of man the iso-electric point shifts to the region of p_H 6.2-8. This shift of the iso-electric point toward the alkaline is of importance, since it favors the loss of lens substance that characteristically follows formation of a cataract.

J. E. LEBENSOHN.

Neurology

PROGRESSIVE MYASTHENIA INVOLVING THE SMOOTH MUSCLES OF THE EYE. V. RABINOVITCH and G. VENGRJENOVSKY, Arch. d'opht. **52:** 23 (Jan.) 1935.

Progressive myasthenia has been considered for some time a disease primarily of the striated muscles. A few isolated reports of cases in which smooth muscles have been involved have appeared. The authors describe a case with the purpose of drawing the attention of neurologists and ophthalmologists to the need for a more careful study of the accommodation in this disease. In their case the results were interesting. The patient was a laborer, aged 22. The left eye was able to accommodate to 7 cm. for one minute. After this the near point gradually receded. After eight and a half minutes of reading it was 16 cm.; after nine and a half minutes, 20 cm.; after ten minutes, 30 cm., and after twelve minutes, 40 cm. The same results were obtained with the right eye, except that the punctum proximum was 12 cm. at the beginning of the test. This tiring of the muscles of accommodation is comparable in some ways to the accommodative asthenopia which follows some severe general diseases such as scarlet fever. The participation of smooth muscle in the disease has been explained by Kramer as having a neurovegetative basis. The theoretical considerations relative to this disease are briefly stated.

S. B. MARLOW.

CHIASMATIC ARACHNOIDITIS. JORGE MALIBRAN and MANUEL BALADO, Arch. de oftal. de Buenos Aires **8:** 199 (May) 1933.

After referring to a former case of traumatic arachnoiditis successfully treated by opening the optic nerve sheath, which they reported, the authors report a case of similar etiology in which paleness of the optic nerve disks with rapid loss of sight followed cranial traumatism. The right visual field showed an enormous scotoma at the center and relative scotomas in the remainder of the field with characteristic island formation and central depression, and the left visual field, a depression of its upper part. The roentgen observations were negative. Craniectomy with opening of the thickened, distended sheath of the optic nerve was followed by marked visual improvement. A reduced central scotoma persisted in both eyes.

The scanty literature is reviewed. The ophthalmoscope shows atrophy of the optic nerve without prior neuritis. The diagnosis rests on quantitative perimetry. There are three groups of cases: those with typical chiasmic symptoms, those with a general perimetric depression and those with relative central scotoma. The case reported by the author belongs to the last group. The treatment is purely surgical.

C. E. FINLAY.

Ocular Muscles

THE RESULT OF ORTHOPTIC TREATMENT IN DIVERGENT STRABISMUS. S. MAYOU, Brit. J. Ophth. **19:** 37 (Jan.) 1935.

Of ninety-three patients with divergent strabismus, forty-eight were emmetropic, twenty-six were hyperopic, and seventeen were myopic. The high percentage with emmetropia is noteworthy. Up to the age of 12 years the results of orthoptic treatment are uniformly good. A few of the patients over 20 years of age have developed binocular vision. In some cases seven lessons with home exercises have proved sufficient, but in others it has taken fifty lessons and home work to obtain a satisfactory result.

From the point of view of treatment, cases may be considered under the following heads:

1. Divergent strabismus curable by orthoptic treatment only
   - (*a*) Alternating strabismus with small angle
   - (*b*) Unilateral strabismus with small angle
   - (*c*) Divergent strabismus associated with hyperphoria and cyclophoria
   - (*d*) Periodic or occasional divergent strabismus
2. Divergent strabismus curable only by operation together with orthoptic treatment
   - (*a*) Alternating divergent strabismus with an angle larger than from 10 to 15 degrees, approximately
   - (*b*) Unilateral divergent strabismus with a large angle
   - (*c*) Postoperative divergence

The following conclusions are emphasized: Patients with divergent strabismus benefit from orthoptic treatment whether they can be cured by treatment alone or whether they need an operation. The majority of patients are cured by orthoptic treatment alone without operation. Among patients with squint the percentage of emmetropic patients is very high, and the majority of them may be classified as having periodic or occasional divergent strabismus. There are very few divergent squints associated with amblyopia.

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

SEVERE FATAL OSTEOMYELITIS OF BOTH ORBITS: REPORT OF A CASE. H. PUSCARIU and D. LAZARESCU, Klin. Monatsbl. f. Augenh. **93:** 531 (Oct.) 1934.

The authors give a synopsis of the literature in point and details of the differential diagnosis and etiology. Then they report the case of a child, aged 2½ years, who was admitted to the hospital with bilateral

exophthalmos, which had developed about six months after an attack of measles. The progress of the disease was slow; no cause was known; high fever was absent; the general physical condition had been satisfactory. Increase of the exophthalmos, the only symptom for a long time, suggested inflammation of the tissues of both orbits. Pus was found after opening the ethmoid sinus. Despite this procedure the infection involved the orbital walls, spread into both antrums and caused multiple abscesses containing staphylococci. Death resulted from an accumulation of pus which separated the dura mater from the cranium and pushed the brain into the anterior portion of the skull. A roentgenogram had suggested the presence of a tumor. The brain remained free from infection, as the dura mater formed a wall against the entrance of the infected pus.

K. L. STOLL.

Pharmacology

A HISTOLOGIC STUDY OF THE NORMAL HUMAN EYE UNDER THE INFLUENCE OF ATROPINE AND PILOCARPINE. M. TEULIÈRES and M. J. BEAUVIEUX, Arch. d'opht. **52:** 65 (Feb.) 1935.

The authors had an opportunity to study the histologic changes in the normal eye following instillation of atropine and pilocarpine hydrochloride. The instillations were made ten minutes after death, the effect being noticeable in ten minutes and complete in twenty minutes. The eyes were enucleated about an hour after death. It was thus possible to compare sections of two eyes of the same person. The report is strictly limited to the histologic description of these two eyes, no reference being made to the literature on this subject. A description of the gross changes which result from the use of these drugs is followed by a detailed histologic discussion, illustrated by some excellent photomicrographs in color. The changes produced in the iris and ciliary body are separately described, but no consideration is given to any variations in the zonule and lens, owing to the difficulties of embedding and cutting sections which will be free from artefact. The antagonistic action of the sphincter and dilator of the iris and the concomitant changes in the iris are shown. In discussing the ciliary muscle the authors state their belief that the sections show that the whole muscle is relaxed under the influence of atropine and that the whole muscle contracts under the influence of pilocarpine. The conclusion, therefore, is that there are no antagonistic elements in this muscle. In reviewing the changes in the iris angle and the spaces of Fontana the authors point out that while atropine narrows the normal avenues of drainage it does not close them. They, therefore, think that the variations in intra-ocular tension arising from the use of atropine and pilocarpine may be more vascular than mechanical.

S. B. MARLOW.

MIOTIC AND ANTIEXOPHTHALMIC ACTION OF CORYNANTHINE. L. JUSTIN-BESANÇON, D. BOVET and D. KOHLER, Compt. rend. Soc. de biol. **118:** 532, 1935.

Corynanthine is the levogyric isomer of yohimbine and, though less toxic, is a more active sympatholytic drug. Moreover, corynanthine possesses only a purely miotic action, whereas the miosis produced by

yohimbine is transitory and is succeeded by mydriasis. In the anesthetized dog, corynanthine chlorhydrate injected intravenously (2 mg. per kilogram) counteracts the exophthalmos produced by ephedrine intravenously injected (5 mg. per kilogram). The pharmacology of the drug suggests that it may be of value clinically.

J. E. LEBENSOHN.

LOCAL ANESTHETIC ACTION OF CORYNANTHINE. RAYMOND-HAMET, Compt. rend. Soc. de biol. **118:** 774, 1935.

Corynanthine, the levogyric isomer of yohimbine, though more potent than the latter in its systemic sympatholytic action, is a much feebler local anesthetic. Tested on the rabbit's cornea—the lid-closure reflex being used as a criterion—the anesthetic action of 0.5 per cent yohimbine is equal to that of 1 per cent cocaine, whereas the anesthetic action of 1 per cent corynanthine is no more than that of 0.25 per cent cocaine.

J. E. LEBENSOHN.

Physiology

INFLUENCE OF THE RETINAL AREA ON THE CRITICAL FREQUENCIES OF LIGHT FLASHES. H. PIERON, Compt. rend. Soc. de biol. **118:** 25, 1935.

Since the fovea represents a region of functional homogeneity, stimulation was made with light surfaces the images of which fell within the foveal confines. At an illumination of 2 millicandles per square centimeter the interval between stimuli at the fusion point (when the light stimulus appeared continuous) was 15.6 sigma for a surface of 30 seconds' diameter (width of a cone) and increased progressively to 39.5 sigma for a surface with a diameter of 24 minutes and 20 seconds (about half the foveal diameter). The interval decreased proportionately with increasing brilliance of the light stimuli.

J. E. LEBENSOHN.

Retina and Optic Nerve

SOME PRACTICAL POINTS IN THE TREATMENT OF SIMPLE DETACHMENT OF THE RETINA. HAROLD RIDLEY, Brit. J. Ophth. **19:** 101 (Feb.) 1935.

It is possible to abstract only a few of the diagnostic helps in Ridley's suggestive paper. In high myopia the tear is apt to be large and situated in the upper half of the retina, some distance from the periphery. In emmetropia and hyperopia there is likely to be a disinsertion in the lower temporal quadrant. Flashes of light in the lower nasal field followed by a shadow beginning in the same situation are almost pathognomonic of a hole in the upper temporal quadrant. In cases of direct trauma the tear may be found in the quadrant where the blow was received. Long-continued loss of the upper half of the field is almost certain to be associated with a disinsertion and not with a true tear. Retinal cysts generally occur in the upper temporal quadrant in persons

of middle age. They are noticed suddenly. The detached retina looks thin and transparent. No hole is found. These cysts are present in about 3 per cent of the cases.

In orienting the hole by the clock face it is better to allow 2 mm. for each disk diameter and 10 mm. for the distance between the limbus and the periphery of the retina.

In the operation itself two undertakings are essential: causing a plastic choroiditis in the right region to seal the hole, and draining the fluid. The latter is, if anything, the more important. If the retina fails to fall against the prepared choroid, the effect of the diathermy is wasted. In the application of the Larsson method only the tip of the electrode should touch the patient. The current too often leaks away through the conjunctiva touching the shaft. The electrode should be pressed firmly against the sclera before the current is turned on, and the area should be free from blood but moistened with distilled water, through which the current does not leak, and which prevents charring of the sclera. Applications some distance from the hole join the retina and choroid over a broad area and diminish the risk of secondary tears.

Seventy milliamperes of current applied for seven or eight seconds appears to be most effective. Trephining the sclera has generally replaced cautery for drainage. Drainage must take place from an area of deep detachment, which must be kept dependent after the operation.

Eyes operated on successfully show, in the first few days, white exudates superficial to the retina. On top of these a small hemorrhage is quite frequently seen. Most eyes worth operating on once are worth operating on a second time if the hole remains unclosed.

W. ZENTMAYER.

POSTPARTUM AMAUROSIS: CURE BY LUMBAR PUNCTURE. VILLARD, VIALLEFONT and DIACONO, Arch. Soc. d. sc. méd. et biol. de Montpellier **15:** 365, 1934.

Fifteen days after normal delivery of her second child, a woman of 23 began to have headache, periorbital pain, bilateral amblyopia and mild bilateral papilledema. After a diagnostic lumbar puncture a marked improvement in vision followed; and after a second lumbar puncture three weeks later, vision in each eye steadily improved until it was normal. No cause for the disturbance was discovered, as all tests of renal, vascular and humoral functions were essentially negative.

J. E. LEBENSOHN.

CLINICAL AND HISTOLOGIC STUDY OF RETRORETINAL HEMORRHAGE. M. CORRADO, Ann. di ottal. e clin. ocul. **62:** 1009 (Dec.) 1934.

A woman of 68 was seen in 1931 with senile macular degeneration of one eye. Two years later vision was reduced to perception of hand movements with this eye, and the retina to the nasal side of the disk was elevated. It was immovable and of a brownish slate color. There was marked arteriosclerosis, and the blood pressure was 210 systolic and 90 diastolic. Transillumination showed a shadow corresponding

to the lower part of the globe. A tumor was suspected; retroretinal hemorrhage was also considered possible, as there were small hemorrhages to be seen at the edge of the detachment. The eye was enucleated and showed a hemorrhage under the detached retina. Marked cystic degeneration of the retina was also present. The rods and cones over the hemorrhage were absent or much degenerated. The pigmented epithelium remained adherent to the detached retina, indicating that the hemorrhage had come from one of the choroidal vessels.

The differential diagnosis between tumor and retroretinal hemorrhage is considered. Corrado concludes that diagnostic puncture is not certain enough to justify the danger of dissemination involved in case a tumor is present, and that an absolute diagnosis is not always possible.

S. R. GIFFORD.

CHANGES IN THE PIGMENTED CELLS OF THE RETINA IN HIGH MYOPIA AND THEIR IMPORTANCE IN THE PATHOGENESIS OF RETINAL DETACHMENT. D. GIANINNI, Ann. di ottal. e clin. ocul. **62**: 1024 (Dec.) 1934.

Gianinni reviews the retinal changes in cases of detachment, especially in cases in which the condition was associated with myopia and advanced age, which may be considered as predisposing to retinal detachment. Redslob, Fuchs and others found retinal tears in most eyes examined histologically. In four of twelve cases no hole could be discovered. Cystic degeneration of the periphery was found in most eyes and after a marked atrophy of the retina. Sourdille believed that disturbances in embryonic life prevent the normal union between the outer and inner layers of the optic vesicle, predisposing to later separation between the pigmented epithelium and the inner layers of the retina. Bartels places more importance on cystic degeneration in the retina, the fluid finding its way to the deeper layers and separating them from the pigmented epithelium.

Gianinni examined a number of eyes histologically, especially those with high myopia, paying special attention to the pigmented epithelium and its relation to the rods and cones. In such eyes the pigmented epithelium was seldom normal. The cells were often swollen and had lost their sharp outlines, while in the cells the pigment was irregularly clumped, the granules losing their characteristic rod shape and becoming round and swollen. At times the layer of pigmented epithelium split into two layers, and often only a few free granules of pigment were left in the lamina vitrea. The retina over such areas showed degenerative changes and often contained pigment granules. Histologic preparations were useful in the study of the pigmented epithelium. The choroid beneath showed marked atrophy, the choriocapillaries being largely absent. Such changes were most marked near the equator and around the optic disk. There were some areas, however, in which the choroid was unchanged while the pigmented epithelium showed the changes described. Gianinni believes such changes must be considered among the factors predisposing to retinal detachment.

S. R. GIFFORD.

ELECTROLYSIS IN THE TREATMENT OF DETACHMENT OF THE RETINA: REPORT OF EIGHTEEN CASES. A. VON SZILY and H. MACHEMER, Klin. Monatsbl. f. Augenh. **93**: 721 (Dec.) 1934.

The authors render a detailed account of their clinical experiences with bipolar electrolysis in the treatment of detachment of the retina. The method is described, also an instrument with exchangeable electrodes, for either perforating or surface electrolysis; it was devised by the authors. Illustrations of the parts of this instrument and pictures of the fundus before and after the operation and of the fields of vision are added; tables show the properties of the detachments, the ages of the patients and the results of the treatment.

Eighteen cases are reported; in two the detachments were treated with perforating electrolysis and in the remaining sixteen with surface electrolysis. The clinical results confirmed those of the experiments on rabbits' eyes published by the same authors (*Klin. Monatsbl. f. Augenh.* 92: 44 [Jan.] 1934; abstr., *Arch. f. Ophth.* **13**: 113 [Jan.] 1935). The advantages of the bipolar galvanic current consist in the certainty of obtaining localized action and in the possibility of knowing the strength of the current, so that lesions can be avoided. The scars are fine, subtle and even, as demonstrated in several pictures. It is the property of the galvanic method to produce chemically an albuminous subretinal exudate which furthers reattachment. The method is simple and may be modified to suit various employments. A definite evaluation of this method with reference to other methods cannot be given. The difficulties met with in any operative procedure in the treatment of detached retina rest finally in the nature of the disease. The simplicity and the reliability of the electrolytic method, however, seem to eliminate many technical difficulties, thus rendering it more practical for the use of the oculist.

K. L. STOLL.

RETINAL DETACHMENT IN RELATION TO AGE. K. VOM HOFE, Klin. Monatsbl. f. Augenh. **93**: 745 (Dec.) 1934.

Leber pointed out the relation between detachment of the retina and the age of the patient; it occurs at any age, rarely before the twentieth, most frequently between the fiftieth and sixtieth years. The frequency of complicating myopia was referred to by several authors.

Vom Hofe reports on 100 patients with detached retina examined at the eye clinic of the University of Cologne during the past two years. After elimination of the cases of traumatic origin, he found myopia of over 7 diopters in 35 patients. In a second group of 43 patients 7 had myopia of 6 diopters or less, whereas the others had no myopia. The number of the patients with detached retina increased with each decade, no matter whether high myopia was present or not. This frequency rose gradually up to the fiftieth year and rapidly after that age. Vom Hofe agrees with Vogt, who stated that the age and the myopia are of equal importance in this respect.

Discussing the etiology of detachment of the retina in young emmetropic patients, vom Hofe refers to pathologic changes such as anemia and bluish sclera. In his opinion, therefore, congenital or localized

inferiority of the retinal tissue may be the etiologic factor. This theory seems to be supported by the case of a healthy and emmetropic man, aged 34, who had binocular detachment of the retina with symmetrically located tears in the temporal and lower areas of the fundus of both eyes.

K. L. STOLL.

PSEUDOXANTHOMA ELASTICUM AND VESSEL-LIKE STREAKS IN THE FUNDUS. A. MATRAS, Wien. klin. Wchnschr. **48:** 198 (Feb. 15) 1935.

Matras reviews the literature on this disease and shows that two types exist: the one with at least half of the criteria outlined by Arzt, Friedmann, Gans and Darier, the other with lesions around the face and infrequently lesions in the skin of other portions of the body.

Four patients are described in detail, the description including a histologic report. In each patient the ophthalmologist Bock found angioid streaks in the retinas. Matras' conclusions based on these observations endorse the theory that the condition is of congenital origin and on a familial basis, as two of the patients were blood relatives and another was the offspring of consanguineous parents. The disease began in early childhood or near puberty.

L. L. MAYER.

THE QUESTION OF POSTOPERATIVE DETACHMENT OF THE RETINA FOLLOWING CYCLODIALYSIS. A. PILLAT, Ztschr. f. Augenh. **85:** 5 (Dec.) 1934.

Pillat reviews the possible damages to an eye subjected to cyclodialysis. He notes that a detachment of the retina may be present as an expression of shrinking of the vitreous or the uvea. This may be attributed to a faulty technic or to a reaction of the tissue to operative trauma. The detachment may be produced by the difference between the curvature of the spatula and the convexity of the tissue. Frequently the detachment is accompanied by hypotony. It may also be stationary.

Pillat describes a case in which there appeared at the ora serrata a small, sharply defined, raised, semicircular area. When the pupil was dilated, the blood vessels were noted on the raised tissue, and the subretinal fluid was clear. No disturbance of vision was noted, and the visual fields were not disturbed. Operative intervention led to reattachment of the retina, and the result was favorable. Pillat warns that this type of detachment of the retina may frequently follow cyclodialysis.

A. M. YUDKIN.

EXPERIMENTAL INVESTIGATIONS ON NYCTALOPIA IN AXIAL OPTIC NEURITIS. SIMIZU, Acta soc. ophth. jap. **38:** 106 (July) 1934.

Patients suffering from nyctalopia have better vision on contracting the palpebral fissure. If the substances containing vitamin B are excluded from the diet of healthy persons an axial optic neuritis develops, and thus nyctalopia can be produced in a previously healthy person. The peripheric visual field shows no change. The pupil of the nyctalopic eye is somewhat smaller than normal. Simizu believes that

nyctalopia in axial optic neuritis is due to pathologic changes of the retina or of the optic nerve. The symptoms of nyctalopia persist for some time even though they are no longer marked.

A. KNAPP.

Trachoma

LYSOZYME CONTENT OF TEARS IN TRACHOMA. A. N. MOURZINN and E. G. SOUCHKOVA, Rev. internat. du trachome **12:** 1 (Jan.) 1935.

All the significant investigations on lysozyme since Flemming's discovery of it in 1922 are carefully reviewed. In acute conjunctivitis the lysozyme values are diminished, and in xerophthalmia they are particularly low. The authors compare the lysozyme content of tears in sixty-four cases of trachoma in various stages with the content in tears of normal eyes and of those affected by diverse maladies. In trachoma the lysozyme content definitely tends to diminish, and the loss is greatest in the primary stages and in the grave cases with corneal ulceration, while in the end-stages of the disease the value approaches normal. The titer is not affected by the age or sex of the patient or by the use of atropine.

J. E. LEBENSOHN.

THE ONLY EFFICIENT PROPHYLAXIS OF THE SOCIAL SCOURGE, TRACHOMA. TALBOT, Rev. internat. du trachome **12:** 15 (Jan.) 1935.

Trachoma is a contagious infection and is transmitted most often by direct contact. Generally trachoma in adults is but a reactivation of a latent primary infantile infection. Children usually acquire the disease before the school age from their nurse or from contact with infected children. Infection at school is exceptional. A regression of endemic trachoma can be secured only through the general treatment and cure of latent infantile trachoma. Any other program must prove illusory.

J. E. LEBENSOHN.

PTERYGIUM AND TRACHOMA. M. E. ALVARO, Rev. internat. du trachome **12:** 32 (Jan.) 1935.

In the clinic at São Paulo, Brazil, the frequency of pterygium among all persons with diseases of the eye is 5 per cent, whereas there is but half this incidence among the trachomatous ones. Possibly the photophobia of the latter protects their eyes from the irritative action of dust and wind.

J. E. LEBENSOHN.

TREATMENT OF TRACHOMA WITH GINGER. J. N. SOLOTNITZKY, Rev. internat. du trachome **12:** 34 (Jan.) 1935.

A thorough mixture is made of 1 part of powdered ginger root with 3 parts of powdered confectionary sugar. This is applied freely to the everted lids; the eyes are then closed for five minutes; at the end of this period the lids are reeverted and thoroughly irrigated. The treatment is particularly efficacious in trachoma with pannus, with or without

accompanying corneal ulceration. The author treated fifty patients in this way and is enthusiastic over his results. Photophobia, lacrimation and blepharospasm generally disappear in from two to three days.

J. E. LEBENSOHN.

Tumors

ORBITAL NEOPLASM OF DIFFICULT DIAGNOSIS. V. MORAX and F. ROUSSEAU, Ann. d'ocul. **172:** 41 (Jan.) 1935.

A young girl of 11 years, following a slight infectious condition considered to be influenza, started to show an inflammatory process in the left orbital region, with swelling of the upper lid, lowering of the left globe and a palpable new growth. Progressively and slowly the tumor invaded the whole orbit, and exophthalmos became so severe that the globe was completely immobile. About this time there appeared on the cornea erosions which caused great irritation and photophobia.

About one year later, as none of the treatments employed had arrested the progress of the lesion, the contents of the orbit were removed. The new growth was adherent on one side to the bony orbit, and on the other, to the muscles and posterior part of the globe. By diffuse infiltration it had penetrated the muscles, superficial layers of the sclera and adipose and other tissues. On account of its extreme rarity sections were submitted to Redslob, of Strasbourg, who replied that he had never seen anything like it and did not possess any similar specimen in his collection. Regaud examined the sections and could not pronounce on them. Because of the particularly large number of eosinophil cells, Morax thought it was perhaps due to the action of a mycelium. The mycelium network, however, did not correspond to that in sporotrichosis, actinomycosis or streptotrichosis.

S. H. MCKEE.

PRIMARY SARCOMA OF THE INFERIOR OBLIQUE MUSCLE. D. MICHAÏL, Arch. d'opht. **51:** 755 (Dec.) 1934.

A primary tumor of a muscle is rare; one of a muscle of the eye is very exceptional. Michaïl reports a case in which a tumor was found in the inferior oblique muscle of the left eye in a child 10 years old. It was excised and found to be a large round cell sarcoma which had completely replaced the muscle in the affected part and was spreading through the muscle. Such a tumor is of very rare occurrence in the orbit. Michaïl reviews the reports in the literature; only six undoubted cases have been recorded so far, including his own. In three of these six cases the superior rectus muscle was involved; in two, the internal rectus, and in one, the inferior oblique. Two occurred in children 2 and 10 years of age; four in adults. In four cases the course was rapid (from one to three months); in two chronic (from one to three years). In no case has there been recurrence. In three cases the involved muscle was extirpated; in two cases the orbit was wholly exenterated and in one there was a subtotal exenteration. The reports in the literature are listed.

S. B. MARLOW.

CARCIMOMA OF A SEBACEOUS GLAND; THE ACID NUCLEUS OF THE CARCINOMA CELL. TAKAWAZA, Acta soc. ophth. jap. **38:** 158 (Dec.) 1934.

A woman 65 years of age presented a round tumor as large as a walnut in the right upper eyelid. This had existed for three years. It began as an ordinary stye. Histologic examination showed it to be a typical sebaceous carcinoma. There were polymorphic cells with absence of basal membrane, many karyokinetic figures, infiltrative growth, and fat in the tumor cells and tumor paths. There were several acini with uniform cells and chalazion-like tissue. The acid nuclei were irregularly distributed in the cell nests of the tumor. There were some inflammatory infiltrations. The examination showed that the carcinoma developed out of a malignant degeneration of an adenoma.

A. KNAPP.

OGUCHI'S DISEASE. KAWABATA, Acta soc. ophth. jap. **39:** 7 (Jan.) 1935.

Two cases of Oguchi's disease were observed which showed that it is characterized by congenital and stationary night blindness. In one case the parents were related; in the other there was a refractive anomaly. The color sense was normal. The visual field in bright daylight was normal, and in poor illumination, concentrically contracted. Both eyegrounds showed the usual changes of Oguchi's disease. The Mizuo phenomenon occurred after from two and one-half to three hours in a dark room and disappeared after from twenty to fifty minutes in a bright room. On the onset of the Mizuo phenomenon, the light sense in one patient became normal, while in the other it remained unchanged.

A. KNAPP.

CHRONIC RETROBULBAR NEURITIS. SOGA, Acta soc. ophth. jap. **39:** 9 (Jan.) 1935.

Seven cases of chronic retrobulbar neuritis were carefully studied in order to determine any association with avitaminosis. The condition is sometimes known as beriberi amblyopia. In one case, after the patient had been treated with large doses of vitamin B for four weeks superficial punctate keratitis developed in one of the eyes. In another case, a woman 30 years of age presented Bitot's spots in both eyes. After treatment with vitamin A these spots disappeared. Two cases of typical beriberi amblyopia are also described. The patients not treated with vitamin B did just as well as those treated with it. Soga therefore believes that the much heralded success of the treatment with vitamin B is still to be carefully considered.

A. KNAPP.

Uvea

UNUSUAL SPOTS ON THE ANTERIOR SURFACE OF THE IRIS IN NEUROFIBROMATOSIS. SAKURAI, Acta soc. ophth. jap. **39:** 11 (Jan.) 1935.

Two women aged 41 and 32, respectively, and a man aged 43 all suffered from neurofibromatosis. The anterior surface of the iris presented curious multiple small round spots about the size of the head of

a pin and somewhat paler than the surrounding iris tissue. Sakurai believes that these spots are like the other lesions seen in neurofibromatosis.

A. KNAPP.

Visual Tracts and Fields

TRAUMATIC HEMIANOPIA. M. FLEDELIUS, Arch. d'opht. **51**: 561 (Sept.) 1934.

Fledelius points out that whereas patients suffering from severe injuries of the head are in such a condition generally as to make testing the acuteness of vision and the fields of vision impossible, those who have had less severe injuries not infrequently may have lesions of the visual pathways which not only are unnoticed by the patient but also escape the observation of the physician because he examines the fields of vision in a small number of cases only or some time after the injury, when severe visual symptoms have developed. He refers to the important part that wounds of the head sustained in the World War played in the localization of the visual cortex. He divides the subject of traumatic hemianopia into two parts, dealing first with injuries of the occiput and the back of the head and second with those which involve the base, the anterior and the anterolateral portions of the cranium. In reviewing first the diagnosis of posterior injuries he calls attention to the statement that hemianopia occurs in 5 per cent of injuries to the head, whereas fracture in the posterior part of the cranium is found in two thirds of the cases presenting hemianopia. The differential diagnosis between hemianopia due to injury of the occipital lobe and that due to injury of the optic tract is discussed. Other symptoms which may be present are enumerated. Fractures of the base of the skull are often accompanied by ocular symptoms. Coppez' ingenious study of the mechanism of these lesions is reviewed. The value and limitations of roentgen examination are pointed out. Fledelius reports four cases. The first was one of bitemporal hemianopia with evidence of dilatation of the sella turcica. The question of pituitary tumor is discussed in relation to the accident. In the second case a left homonymous hemianopia was associated with signs of involvement of the pyramidal tract. The differential diagnosis lay between an organized hematoma in the right occipital region or a subcortical scar. In the third case there was a right hemiachromatopsia combined with vague mental symptoms which seemed to indicate a lesion of the left occipital lobe due to a subdural hemorrhage. The fourth case is presented because the lesion of the visual apparatus, although of considerable extent, was not discovered until probably fifty years after its occurrence. During early childhood the patient had sustained a severe cranial injury which resulted in deformities of the skull demonstrable by palpation and by x-rays. There were marked optic atrophy on the right side and a suggestion of atrophy on the left. Right homonymous hemianopia was found. The patient had had epileptic convulsions at long intervals since the age of 15.

S. B. MARLOW.

CAUSES OF BITEMPORAL HEMIANOPIA IN PREGNANCY. FRANZ FISCHER, Ztschr. f. Augenh. **85:** 88 (Jan.) 1935.

Fischer analyzes manifestations of bitemporal hemianopia frequently found accompanying pregnancy. Two cases of this disturbance are described.

In the first instance the bitemporal hemianopia disappeared after the pregnancy was terminated. In the second, the pregnancy was terminated before term, and the bitemporal hemianopia improved temporarily. Symptoms of a hypophyseal tumor, acromegalia and dystrophia adiposa genitalis were not observed in these cases. Photographs of the sella turcica showed no abnormalities. The bitemporal hemianopia was therefore attributed to hypertrophy of the hypophysis. Three quarters of a year later the bitemporal hemianopia reappeared, and the visual fields quickly diminished. The symptoms became worse, and a diagnosis of tumor in the region of the optic chiasm was made. At autopsy the diagnosis was verified. The second case likewise showed signs of increased intracranial pressure after the termination of the pregnancy. An operation revealed a tumor mass in the area of the chiasm. Fischer recommends that in all cases of pregnancy in which bitemporal hemianopia is noted, an effort should be made to rule out the presence of a tumor in or about the chiasm.

A. M. YUDKIN.

Sympathetic Ophthalmia

COMPARATIVE STUDIES OF SYMPATHETIC OPHTHALMIA AND ACUTE DIFFUSE CHOROIDITIS (HARADA): II. THE HISTOLOGIC EXAMINATION OF SYMPATHETIC OPHTHALMIA. C. HAMADA, Acta soc. ophth. jap. **38:** 107 (July) 1934.

Six cases of sympathetic ophthalmia were examined and the following conclusions reached:

With destruction of the chromatophores the cell infiltration became more distinct and was situated mostly at the tip of the iris, in the middle of the ciliary body and in the posterior half of the choroid, the regions in which the vascular network is most in evidence.

After the destruction of the chromatophores the pigment granules were scattered between the infiltrating cells and were taken up by the epithelioid and giant cells. The exudate under the retina was most marked about the disk, at the macula and in the equatorial regions and was followed by a typical retinal detachment. The infiltrating cells were also found in the anterior layers of the iris, which is opposed to Fuchs' description. Giant cells were frequently found in the ciliary body. The suprachoroidal space contained a collection of exudate, but the new connective tissue formation which Asayama has described was not found. It was not possible to determine from which cells the epithelioid cells were derived, though there seemed to be a definite relationship between the epithelioid cells and the blood vessels.

A. KNAPP.

COMPARATIVE STUDIES OF SYMPATHETIC OPHTHALMIA AND ACUTE DIFFUSE CHOROIDITIS (HARADA): IV. THE HISTOLOGIC EXAMINATION OF ACUTE DIFFUSE CHOROIDITIS AND GENERAL DEDUCTIONS (HARADA). C. HAMADA, Acta soc. ophth. jap. **38:** 127 (Sept.) 1934.

Hamada has compared the various clinical symptoms in twenty cases of sympathetic and diffuse choroiditis (Harada), adding two new cases of acute diffuse choroiditis with severe iridocyclitis and one case of idiopathic uveitis (Vogt), also two cases of acute diffuse choroiditis occurring many years after an injury to the eye. Three of these cases were examined histologically. The results were as follows:

1. Except for the injury of one eye the ocular and general symptoms of sympathetic ophthalmia with relatively mild iridocyclitis resembled acute diffuse choroiditis (Harada). The symptoms of the idiopathic uveitis (Vogt) and of the acute diffuse choroiditis with severe iridocyclitis resembled those of the sympathetic ophthalmia with severe iridocyclitis. The histologic observations were the same.

2. If the uveitis which develops after a certain length of time following an injury is called sympathetic ophthalmia, the peculiar uveitis which occurs much later after an injury without any fresh cause should be called acute diffuse choroiditis.

3. Both diseases have an acute onset and present exudative changes and toxic manifestations. In addition to the uveal system, the pigment cells in the rest of the body are affected, in the meninges, vestibule, cochlea, hair and skin.

4. The proliferative changes in the uvea are accompanied by gradual destruction of the chromatophores. The pigment granules are to be found in the epithelioid and giant cells. These proliferative and infiltrative changes as well as the atrophic changes are secondary to toxic manifestations.

5. All these findings are explained by Nakamura's toxine theory, which shows that the serum of guinea-pigs immunized against rabbit's uvea is extremely toxic to rabbits which have undergone an injury to the uvea of one eye two or three weeks previously. The action on the normal rabbit is much less toxic. Sympathetic ophthalmia corresponds to the first case, while acute diffuse choroiditis corresponds to the second.

A. KNAPP.

Therapeutics

TECHNIC AND EFFECT OF INJECTION OF AUTOGENOUS BLOOD INTO THE ANTERIOR CHAMBER (ACCORDING TO SCHIECK) IN TUBERCULAR IRIDOCYCLITIS. W. KYRIELEIS, Ztschr. f. Augenh. **85:** 16 (Dec.) 1934.

The injection of autogenous blood into the anterior chamber of the eye in tuberculous iridocyclitis is beneficial. The improvement of vision parallels the diminution of deposits on the cornea. Kyrieleis asserts that the deposits on the posterior layer of the cornea are broken down by the mechanical movement of the blood elements in the anterior cham-

ber. Local irritation and increased hyperemia are produced, which affords an absorption of the newly broken-down pathologic tissue. Antibodies are introduced by this process into the anterior chamber. Tubercular nodules are frequently disseminated and disappear. Kyrieleis offers this treatment as an adjunct to other therapy in tuberculosis. The treatment is contraindicated in the acute exudative stage of iridocyclitis (tuberculous) and when new nodules are being formed.

A. M. YUDKIN.

PERCUTANEOUS TUBERCULIN TREATMENT OF OCULAR DISEASES BY SCARIFICATION WITH GLASS OR EMERY PAPER. A. LÖWENSTEIN, Ztschr. f. Augenh. **85:** 191 (Feb.) 1935.

Disappointed with the previous methods of tuberculin treatment, Löwenstein proposes the following procedure for the treatment of tuberculosis of the eye. The upper layer of the skin is scarified with a glass slide or with emery paper so that no bleeding is produced. He suggests that the skin on the outer portion of the upper arms, shoulders or thoracic wall, where the hair is sparse, be used for this purpose. In the abrased area increasing doses of from 1 to 8 drops of concentrated old tuberculin are rubbed in with a glass rod. The manipulation is painless, and a child will not resent the application. A specific reaction is manifested by a diffuse local erythema in twenty-four hours. Blisters may form, but they gradually dry up. The most aggravated reaction disappears in from eight to ten days. Löwenstein has never observed an increase of temperature during this therapy. At the height of the reaction, headache may be present. A positive reaction is manifested by focal reaction. To date this procedure has proved to be very beneficial in tuberculous conditions of the eye.

A. M. YUDKIN.

Book Reviews

Bulletin of the Ophthalmological Society of Egypt. Volume 27. Pp. 145, with 20 plates. Cairo: Misr Press, 1934.

The thirty-first session of the Ophthalmological Society of Egypt was held on March 23, 1934, at the Giza Memorial Ophthalmic Hospital, with Dr. Mohammad Aziz Barrada presiding.

Dr. A. M. Sulliman, who had recently visited the European clinics, described Sinclair's and Elschnig's intracapsular cataract operations; the orthoptic treatment of squint with the synoptophore; iontophoresis in cases of glaucoma, using an epinephrine substitute; sclerectomy with a trephine in performing cyclodialysis, and Weve's apparatus for localization of the tear in detachment of the retina.

Dr. Barrada described operations for detachment as performed by Guist, Lindner, Krückmann, Šafář, Larsson and Coppez, and Dr. A. Y. Fahmy described a combined operation for total detachment in which scleral resection was done, with application of potassium hydroxide to the exposed choroid. Dr. M. Sobhy Bey also made a few general remarks on the same subject.

The pathology of dacryocystitis was described, with the showing of twenty-four microphotographs, by Dr. A. Y. Fahmy, and Dr. Max Meyerhof reported two cases of secondary follicular conjunctivitis occurring on an old trachoma. Dr. Barrada also read papers on subhyaloid exudation, avascular keratitis and argyrosis of the conjunctiva, a filaria in the macula, herpes zoster ophthalmicus and a case of ophthalmomalacia (essential), written in collaboration with Dr. M. A. Bakly, and Dr. M. Riad described the clinical findings in cases of ocular leprosy.

In the section on therapeutics Dr. Nessim Abu-Saif reported favorable results in cases of blepharitis in which epilation of the lashes was obtained with the roentgen rays. After epilation occurred, either ointment of yellow mercuric oxide or ammoniated mercury ointment was used locally. Twenty patients were treated; there were two relapses.

Dr. Fayek Fahim reported a case of idiosyncrasy to both yellow oxide and zinc sulphate ointment and also reported on the favorable visual results obtained by treating corneal opacities (none of which was of longer duration than eight months) with subconjunctival injections of from 1 to 5 per cent solution of ethylmorphine hydrochloride, and Dr. Amin Migally reported on the value of saturated solution of a magnesium sulphate in the treatment of acute inflammations of the conjunctiva and cornea.

There were also two papers in French: one by Professor Salvati, on the etiology of preretinal hemorrhage, which he thinks is often due to hypertension, and one by Dr. J. Démétriades on three cases of optic neuritis which occurred after the use of acetylarsen (diethylamine-oxyacethylamino phenylarsinate).

The bulletin ends with a short account of the career of Dr. Max Meyerhof, who was the guest of honor at a dinner given on the occasion of his sixtieth birthday on the evening of the annual meeting.

Dr. Meyerhof, who was president of the Ophthalmological Society of Egypt in 1909, started his study of ophthalmology in 1899. He went to Cairo in 1903, where he has since been engaged in his work except for the years between 1914 and 1922. In addition to writing numerous papers dealing with contemporary ophthalmic problems, he has written extensively on the history of Arabian medicine and ophthalmology and has acquired almost universal recognition as a brilliant scholar of the oriental languages (Hebrew, Syrian, Arabian, Persian and Turkish).

The bulletin also contains a list of the members of the society, the rules of the society and a partial glossary of Arabic ophthalmic terms. There is also a short index of authors and subjects.

W. F. DUGGAN.

Aids to Ophthalmology. By N. Bishop Harman. Eighth edition. Price, $1.25. Pp. 242, with 203 illustrations. Baltimore: William Wood & Company, 1935.

This pocket-size volume is one of the Students' Aids Series, which is intended to assist the student in preparing for examinations as well as to aid the general practitioner in reviewing the various subdivisions of medicine and the basic sciences. The fact that it is appearing in a new and revised edition attests to its popularity in Great Britain. It contains a large amount of fundamental information, which is permitted by the small but clear type in which it is printed.

There are forty chapters, including, in addition to the usual systematic anatomic grouping of diseases of the eye, seven chapters on refraction, which are admirable examples of terseness and completeness. Other chapters deal with the ocular signs of general diseases, ocular therapeutics, operations, ocular conditions found in school children and the various standards of vision required for sundry vocations in Great Britain. There is a good index.

As a compendium of basic and practical ophthalmology, the book merits a continuation of its popularity.

W. F. DUGGAN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St. Honoré, Paris.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.
Place: Cairo. Time: 1937.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. A. J. Ballantyne, 11, Sandyford Pl., Glasgow, C 3.
Secretary: Dr. J. Ringland Anderson, 108 Collins St., Melbourne.
Place: Melborurne. Time: Sept. 11-13, 1935.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. R. Foster Moore, 53, Harley St., London, W. I.
Secretary: Miss Ida Mann, 7, Wimpole St., London, W. I.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Royal Society of Medicine, Section of Ophthalmology

President. Dr. Ransom Pickard 31, East Southernhay, Exeter, England.
Secretary: Dr. J. D. Magor Cardell, 27, Weymouth St., London, W. 1.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.

Chinese Ophthalmological Society of Peiping

President: Dr. P. H. Liu, Chi-Ming Eye Clinic, Ta Liu Pu Kau, Peiping.
Secretary: Dr. H. H. Feng, Peiping Union Medical College, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. John Green, 3720 Washington Blvd., St. Louis.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Kansas City, Mo.: Time: May 11-15, 1936.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
President-Elect: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Cincinnati. Time: Sept. 16-20, 1935.

American Ophthalmological Society

President: Dr. Louis S. Greene, Washington, D. C.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Hot Springs, Va. Time: June 1-3, 1936.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

New England Ophthalmological Society

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. H. G. Merrill, 3245 4th St., San Diego, Calif.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Del Monte, Calif. Time: Spring, 1936.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Francis A. Brugman, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Noble Dick, 1115 Terry Ave., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. W. R. Fringer, 228 S. Main St., Rockford, Ill.
Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.
Place: St. Louis. Time: November 1935.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. F. B. Fralich, 202 S. Main St., Ann Arbor, Mich.
Secretary-Treasurer: Dr. A. S. Barr, 201 S. Main St., Ann Arbor, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Claude W. McKee, 229 S. Main St., Greensburg.
Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.
Place: Indiana. Time: October 1935.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227, 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. George B. Garlick, 144 Golden Hill St., Bridgeport.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.
Place: Savannah. Time: May 12-15, 1936.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.
Place: Martinsville. Time: April 8, 1936.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. W. Dean, 532, 1st Ave., Council Bluffs.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Council Bluffs. Time: Nov. 7, 1935.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. John W. Orr, 503 S. Saginaw St., Flint.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. E. Binger, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Place: St. Paul. Time: May 1936.

Montana Academy of Oto-Ophthalmology

President: Dr. George A. Lewis, Roundup.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Place: Billings. Time: February 1936.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. John F. Fairbain, 925 Delaware Ave., Buffalo.
Secretary: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.
Place: Jamestown. Time: May 1936.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. Josiah E. Smith, 60 Meeting St., Charleston.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. William Stookey, 54 E. South Temple St., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welsh, 711 Boston Bldg., St. Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Emory Hill, 501 E. Franklin St., Richmond.
Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.
Place: Richmond. Time: May 1936.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. B. E. Failing, 31 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. Herschel Crawford, 478 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. Stacy C. Howell, 384 Peachtree St., N. E., Atlanta, Ga.
Place: Academy of Medicine, 13 Queen's Park. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John H. Bailey, 855 St. Marks Ave., Brooklyn.
Secretary-Treasurer: Dr. George Freiman, 703 Grand St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.
Time: Second Thursday of each month.

Chicago Ophthalmological Society

President: Dr. Robert B. Blue, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Thomas D. Allen, 122 S. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. F. W. Thomas, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.
Place: Athletic Club. Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. H. Newton, Mercantile Bldg., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St, Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. T. L. Goodman, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. John H. McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 307 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months. September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. R. M. Hargrove, 1202 Esperson Bldg., Houston, Texas.
Secretary: Dr. J. C. Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde E. Harner, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul T. Southgate, 704 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Walter R. Crane, 756 S. Broadway, Los Angeles
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. E. A. Leggett, 614 Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. E. Craddock, 604 Breslin Bldg., Louisville, Ky.
Place: Jewish Hospital. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. J. J. Brook, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6 p. m., second Tuesday of each month.

Montgomery County Medical Society

Chairman: Dr. W. J. Blackburn, 663 Reibold Bldg., Dayton, O.
Secretary-Treasurer: Dr. F. J. Driscoll, 986 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Secretary: Dr. A. L. Guertin, 410 Sherbrooke St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Wesley Wilkerson, Bennie Dillon Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Hale Cullom, Bennie Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LaGrand H. Hardy, 30 E. 40th St.,, New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. John H. Judd, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. A. Cassidy, 107 S. 17th St., Omaha
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, 500 Penn Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT-LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Samuel M. Cottrell, 116 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Laurence Macey, 221 Alexander St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Meyer Wiener, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, Metropolitan Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Frank Steed, Nix Professional Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Secretary: Dr. Louis F. Morrison, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Marc Anthony, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. Raymond A. Lower, 1455 Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Arnold B. Kauffman, 104 W. Onondaga St., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St. W., Toronto.
Secretary: Dr. A. Lloyd Morgan, 86 Bloor St., West, Toronto.
Time: First Monday, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Ernest Sheppard, 927 Farragut Sq., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 7:30 p. m., first Monday in November, January, March and May.